

THE DICTIONARY OF PRACTICAL MEDICINE

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IN THREE VOLUMES

WITH 48 PLATES AND 109 FIGURES IN THE TEXT†

VOL. II

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THE DICTIONARY OF PRACTICAL MEDICINE

VOI II

HEAD INJURIES Injuries of the head owe their chief importance to the effects which may be produced upon the brain either directly, or secondarily from damage to the intracranial blood vessels. When a wound exists or when a fracture extends into the nose or ear so as to produce a communication between the exterior and the interior of the cranium, the additional factor of possible microbic invasion of the brain and membranes is introduced. Thus meningitis, abscess, and septic thrombosis of sinuses may result from any compound fracture of the skull.

The effects of cranial injuries upon the brain are grouped by time-honoured custom under the headings of concussion, contusion, and compression. These states naturally tend to merge into one another. Initial symptoms of concussion may pass off and be succeeded by those of contusion and compression.

Concussion is a definite and easily recognized condition. Its pathology has been much debated and the symptoms have been variously ascribed to molecular disturbance, multiple punctiform hæmorrhages, sudden alteration in cerebrospinal fluid pressure, and cerebral anæmia. It is now generally accepted that the symptoms result from anæmia of the brain suddenly produced by momentary deformation of the skull. In the slightest cases only the highest function, consciousness, may be affected; in more severe cases motion and sensibility also suffer, whilst in the most serious injuries the medullary centres are involved, sometimes to such a degree that recovery does not take place.

The clinical symptoms range in severity

from a momentary loss of consciousness to those of profound disturbances of the cardiac and vaso-motor mechanisms.

Loss of consciousness is the outstanding and invariable symptom of concussion; it may last for a few seconds only, or for many hours. In a case of moderate severity the limbs are flaccid, the skin pale and cold, the breathing shallow, the pulse small and slow, and the pupils dilated. As recovery takes place the symptoms of this initial stage are succeeded by others no less definite. Consciousness returns, but memory of the events immediately preceding the accident is lost; muscular power is regained, the skin flushes, the pulse becomes full, the respiration normal, and the pupils contract; vomiting often occurs, and the patient complains of headache.

If, in addition to the concussion, the brain has suffered **contusion** or laceration, or some vessel important enough to give rise to serious bleeding has been torn, then other symptoms make their appearance. The effects of concussion may merge into those of the more serious injury uninterruptedly, or there may be an intervening period between recovery from the concussion and the onset of further symptoms. Persistent headache, irritability, refusal of food, and sleeplessness are among the general symptoms which indicate laceration of the brain. In addition, there may be focal symptoms pointing to the bruising or laceration of some special area of the cortex, but as a rule the parts of the brain which suffer in this manner in the usual head injuries of civil life are the temporal and frontal poles, lesions of which cause inconspicuous neurological symptoms.

The symptoms of cerebral compression are

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definite and often insistent. The brain is remarkably tolerant of compression so long as it is increased very slowly, but when the rise of pressure is brought about rapidly, as it is by a hæmorrhage, symptoms are quickly produced. These symptoms, again, are both general and local. If the hæmatoma occurs in the most common situation—namely, in the anterior temporo-parietal region—from laceration of the middle meningeal vessels, a progressive contralateral hemiplegia, at first with rigidity, develops. At the same time consciousness is progressively affected, the patient passing through the various phases of stupor from drowsiness to complete coma. As the pressure rises, too, the face becomes dusky flushed from venous stasis, while the same phenomena may be observed in the optic discs, the veins becoming engorged and their edges blurred by oedema. The medullary centres respond characteristically to the increasing intracranial pressure. The pulse becomes slowed; the blood-pressure rises in order to compensate for the increasing difficulty in securing an adequate blood supply for the medulla; the respiration is slow and deep. Unless relief is afforded, this compensatory mechanism breaks down, the cardiac, vasomotor; and respiratory centres, unable to obtain an adequate supply of blood, fail, and death ensues. This medullary failure is heralded by irregularity of cardiac rhythm and Cheyne-Stokes breathing. The pupils are unequal during the advance of the intracranial pressure, owing to the fact that at first the pressure is unequal on the two sides of the falx cerebri. The final state of the pupils is one of wide dilatation and inactivity to light.

TRAUMATIC HÆMORRHAGE

The extravasated blood may be extradural, intradural, or intracerebral. **Extradural hæmorrhage** tends to be limited, so as to form a more or less localized hæmatoma. Its commonest source is the middle meningeal artery, which may be torn at any point from its entrance into the skull onwards. When this vessel is ruptured near the foramen spinosum the resulting hæmorrhage is basal, and the dura is gradually stripped from the bone. As the bleeding progresses the hæmatoma increases in an outward and then an upward direction, following the middle fossa, until it appears beneath the squamous bone. Thus the temporal lobe is directly compressed. In other cases the artery is torn near its point of divi-

sion; then the hæmatoma will be found in the classical situation.

In **intradural hæmorrhage** the blood spreads far and wide over the cerebral surface and mingles freely with the cerebro-spinal fluid. A lumbar puncture will give exit to blood-stained cerebro-spinal fluid, which may be so heavily charged as to resemble pure blood. Occasionally a subdural hæmorrhage becomes definitely localized. The hæmatoma so formed may manifest itself by very slowly progressive symptoms which may only be recognizable some weeks or even months after the original injury. Such hæmatomata take the form of an extensive flattened cyst, the wall consisting of fibrin and the contents of brown fluid. Increasing headache, lethargy, and papilloedema are associated with slowly progressive symptoms of cortical damage; contralateral weakness, sensory loss of cortical type, and hemianopia may all coexist. Such a case must be operated upon without delay. The skull and dura must be opened and the blood cyst removed.

Intracerebral subcortical hæmorrhage may result from a punctured wound when the instrument has missed meningeal and cortical vessels and chanced to wound a more deeply placed artery. Cases of this nature have been recorded after penetrating wounds made by sharp instruments. In cases of gunshot wound hæmorrhages of any considerable size are very rare.

Intracerebral hæmorrhage may occur at almost any time after an injury of the brain, probably in some instances as the result of the giving way of a blood-vessel which has lost its support through traumatic softening of the damaged brain, and in others from rupture of a small traumatic aneurysm. The symptoms closely resemble those of ordinary apoplexy of pathological origin.

Treatment.—A patient who is suffering from simple *concussion* in the early stage should be put to bed and kept warm. In slight cases no special treatment is necessary; in more severe cases stimulants may be indicated by a small, feeble, rapid pulse; headache may call for phenacetin and caffeine; bromides should be given if there is any irritability; aperients should be administered, and a light diet ordered. A period of complete rest, both physical and mental, should be insisted upon, and the length of this period must be determined by the severity of the case, together with the patient's mentality and activities. A business man with

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heavy responsibilities requires a longer period of rest than an unskilled labourer.

Every case of concussion must be carefully watched for the appearance of symptoms of *compression*. Most cases of any severity show some signs of increased intracranial pressure for a few days, as headache, lethargy, slow pulse and engorgement of the retinal veins. These symptoms may be relieved by lumbar puncture but usually no special treatment is called for. When however such pressure symptoms are severe, persistent or progressive and especially if any neurological signs pointing to increasing local disturbance are elicited, relief must be afforded by operation. The site of the opening in the skull must depend upon the localizing signs. But when none are present a right sided subtemporal decompression should be carried out.

FRACTURES OF THE SKULL

1 Fissured fracture. The above mentioned results of a head injury upon the brain may or may not be accompanied by fracture of the cranial bones. Fissured fractures, whether of the vault or of the base are in themselves unimportant. Those of the vault can usually be detected by X-ray examination and at the early stage by some swelling and tenderness along the line of the crack. When the fissure involves the basis cranii so as to pass across the anterior fossa, bleeding may take place from the nose or into the pharynx and cerebro spinal fluid may escape with the blood and continue to flow long after the bleeding has ceased. In rare instances a prolonged cerebro spinal rhinorrhoea results. Fracture of the orbital roof is evidenced by haemorrhage in the orbit and subconjunctival ecchymosis. When the fissure passes through the temporal bones, blood and cerebro spinal fluid may escape from the ears, fracture into the posterior fossa is indicated by subcutaneous ecchymosis which appears over the mastoid processes a few days after the accident.

2 Depressed fracture. When the skull is struck violently by a blunt instrument, or when the patient falls and strikes the head upon some such object as a stone, a localized comminuted fracture with depression of the fragments is apt to be produced. Such a fracture may involve chiefly or even wholly the inner table, so that a depression may exist without any visible or palpable deformity. Even when the bone is exposed by turning down a flap of scalp there may be

no irregularity to be made out, though the site of the injury may perhaps be indicated by a bluish discoloration. Such injuries are of importance in view of the probability that the subjacent cortex has been bruised and that symptoms may subsequently develop.

3 Punctured fracture. Pointed weapons that penetrate the skull produce injuries which are all the more dangerous from the fact that they are septic and that tiny fragments of bone may be driven into the brain substance. Such injuries resemble the type of gunshot wound in which the missile has penetrated but has not remained within the skull.

Treatment. The fissured fracture which so often accompanies the cerebral disturbances resulting from a severe head injury requires no special treatment. Localized comminuted fracture with displacement of fragments, however, demands active measures. When compound the need for operation is the more imperative in order that the dangers of intracranial infection may be minimized. This is particularly true when the dura mater has been lacerated. Simple depressed fractures, also in most, if not in all, cases call for operative treatment, in order that recovery of the damaged brain beneath may not be retarded by continued local pressure. In any case, whether simple or compound, the fracture, which is always comminuted, should be freely exposed by reflecting a large flap of scalp. A trichophene hole should be made close to the edge of the fracture, and the injured bone removed until the operator is satisfied that no depressed fragments remain. The replacement of pieces of bone is of doubtful value, it should never be done in cases of compound fracture. Small lacerations in the dura mater may be closed by the application of a thin layer of muscle or fascia. When a scalp wound exists it should be completely closed by suture after excision of its edges. (See SCALP WOUNDS, TREATMENT OF.)

INJURIES OF CRANIAL NERVES

In fractures of the base of the skull certain of the cranial nerves are liable to be injured, either at their foramina of exit or at some other part of their course. These injuries may take the form of contusion or laceration, and the results may be temporary or permanent. Fortunately, recovery is the rule. The olfactory nerves are apt to suffer when the fracture involves the cribriform plate.

Of the other nerves, the third, sixth, seventh, and eighth are those most commonly affected.

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In this connexion it is interesting to note that the pituitary gland may be damaged in a fracture which traverses the sella turcica, and that glycosuria may result.

INJURIES OF THE VENOUS SINUSES

The *cavernous sinus* may become thrombosed as a result of injury, with consequent proptosis and oculo-motor palsy; such proptosis must be distinguished from that due to orbital hæmorrhage. As an occasional result of coincident injury to the cavernous sinus and the carotid artery, an arterio-venous communication becomes established. This accident is evidenced by the appearance of a "pulsating exophthalmos."

The *longitudinal sinus* may be directly injured, especially in gunshot wounds of the vertex of the skull, or the entering cortical veins may be torn. These injuries give rise to a characteristic group of symptoms, of which the chief are: (a) Spastic weakness of the legs, and sometimes of the arms as well; the spasticity of the legs is most pronounced at the peripheral, and of the arms at the proximal joints. (b) Sensory disturbance of a cortical type. These symptoms tend to spontaneous recovery, and the condition calls for no active treatment unless symptoms of increasing intracranial pressure point to the coexistence of a hæmorrhage.

SEQUELÆ OF CRANIO-CEREBRAL INJURIES

1. *Persistent headache* is a common sequel of these injuries. In many cases it is one of a number of neurasthenic symptoms; in others it is an isolated symptom. The actual causation is obscure, but the relief which may be afforded in a certain number of cases by a simple decompressive operation suggests that a persistent increase of intracranial pressure plays a part in its causation. Many patients who suffer in this manner have had no proper course of treatment after their injury; they have been allowed to get about and follow some occupation too soon. Such patients are greatly benefited by being kept at complete rest in bed for some weeks, whilst at the same time bromides are administered and the general bodily health is attended to.

2. *Epilepsy*.—A small proportion of the subjects of head injury develop fits. Some of these patients are undoubtedly predisposed to epilepsy from hereditary taint. In many instances there exists a definite lesion of some kind, such as a depressed fracture, indriven

bony spicules or a foreign body, adhesions resulting from septic infection, or a blood cyst; but it is clear that post-traumatic fits cannot be ascribed to such lesions alone, seeing that exactly similar conditions are found in a far larger number of patients who never develop epilepsy. Further, in many cases of epilepsy which are ascribed to traumatism no gross lesion can be found.

The actual causal connexion between injury and epilepsy is not fully understood, but it is certain that a gross traumatic lesion is often situated at the part of the brain from which the fits, when they are of a Jacksonian character, originate. It might be expected, therefore, that the removal of such a focus would be followed by cure, but unfortunately experience does not bear out this expectation. Operation almost always modifies the fits in character and frequency, and for months and even years may bring about their complete cessation. Sooner or later, however, relapse is almost sure to occur, more especially if the patient is allowed to resume an active life too soon after the operation. If a patient, after the removal of the supposed epileptogenous focus, is shielded from exertion, both physical and mental, lives a very quiet open-air life, avoids alcohol and every kind of excess, and takes small doses of bromide regularly for a long period, the result may be highly satisfactory. Operation alone cannot cure traumatic epilepsy, but it has a definite place in its treatment.

3. *Neurasthenia*.—A whole train of neurasthenic symptoms may follow a cerebral injury, particularly in patients who have not received a sufficiently careful or prolonged course of treatment after the accident.

4. *Insanity*.—Cerebral injury is rarely the cause of serious mental disorder, though minor, and often only temporary, symptoms such as depression, irritability, and other temperamental changes are not infrequent after serious injuries of the frontal lobes. Except, however, in the case of the grossest lesions, it is doubtful what rôle trauma, apart from hereditary taint, alcohol, and syphilis, plays in the causation of insanity.

5. *Encephalitis*.—The implantation of microbes in the track of a cerebral wound may be followed by any degree of encephalitis, from a diffuse, rapidly spreading septic softening, to a definitely encapsuled abscess which may remain latent for years. In the more acute cases softened brain is protruded through the wound in the form of a fungus cerebri, the cerebro-

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spinal fluid becomes purulent, and death occurs from cerebral compression. In the less acute cases, when an opening exists in the skull and dura mater, a hernia cerebri forms, and persists as long as the intracranial pressure remains above the normal. Thus a hernia cerebri is merely an index of increased intracranial pressure, and by its variations in size and tension constitutes a guide as to whether the infection is spreading or subsiding. The protrusion itself requires no treatment beyond rigid cleanliness and protection; the underlying cause may require surgical intervention, such as drainage, or removal of foreign bodies. Lumbar puncture, or an operation of decompression, may sometimes be employed advantageously for the relief of the intracranial pressure. The late sequelae of gunshot wounds are in all essential respects similar to those which result from injuries of other kinds, and require treatment on similar lines.

PERCY SARGENT.

HEAD-NODDING OF INFANTS (*see* SPASMUS NUTANS).

HEAD'S AREAS (*see* NEURALGIA).

HEALTH RESORTS (*see* CLIMATE IN THERAPEUTICS; HYDROTHERAPY).

HEARING (*see* EAR, EXAMINATION OF).

HEART, CONGENITAL DISEASE OF.

Etiology.—*Developmental errors* are of chief importance. Their occurrence is no more easily explained in the heart than in other parts of the body. There is no family tendency to cardiac malformation, and the claims of the various parental factors which have been suggested—alcoholism, tuberculosis, syphilis, and other forms of ill-health—are based on very imperfect evidence. It is often associated with other defects, among which Mongolian idiocy is perhaps worthy of special mention.

The importance of *intra-uterine endocarditis* has been over-emphasized, and cases so caused form but a small minority.

Pathology.—The cases in which endocarditis is supposed to have been responsible are those in which the mother was attacked by acute rheumatism during pregnancy. In a few of these the cardiac valves of the infant have displayed evidences of recent or active inflammation.

Developmental errors may result from the

interruption of the normal evolution of the heart in the following ways:

(a) The septa which develop centripetally from the walls of the primitive single ventricle and auricle, cutting each of these cavities into two chambers, may be incomplete. In the case of the interventricular septum the defect is most often situated immediately beneath the point of origin of the great vessels, at the spot known as the *pars membranacea septi*, or the undefended space. When the auricular septum is defective the commonest fault is a patency of the foramen ovale—a malformation so common that it scarcely counts as an abnormality.

(b) The cardiac valves may be absent or malformed. By far the commonest example is pulmonary stenosis. The pulmonary semilunar valves may be fused, or, rather, imperfectly separated, so as to form an obstructive collar; or there may be an actual narrowing, amounting in extreme instances to complete atresia, of the arterial tube itself. Sometimes there is an incomplete division of the right ventricle into two chambers by an abnormal constriction situated a little below the pulmonary orifice. The counterparts of these malformations are less often encountered on the left side of the heart in connexion with the origin of the aorta. The error which lies at the root of all these malformations is, according to Keith's view, a failure in the process by which, early in the development of the heart, the bulbus cordis (intervening between the primitive ventricle and the truncus arteriosus) becomes absorbed into the ventricles, and especially the right ventricle. It is not uncommon to find imperfection of the pulmonary artery associated with patency of the interventricular septum at the *pars membranacea*. As a result of this combination of errors the disproportionately large aortic opening is placed just above the defect in the ventricular septum, so that the aorta receives blood not only from the left but also from the right ventricle, the stream from the latter finding easier egress by this abnormal channel than through the narrow and inadequate pulmonary artery. More rarely the great vessels are completely transposed, the left ventricle opening into the pulmonary artery and the right ventricle into the aorta. The mode of origin of this singular malformation is, at present, undetermined. Malformations of the auriculo-ventricular valves are very rare, incomplete division of the tricuspid valves or complete

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atresia of the tricuspid orifice being met with far more frequently than defects of the mitral apparatus.

(c) The great vessels are subject to malformations other than those already mentioned. Of these, two only need be alluded to—patency of the ductus arteriosus, and coarctation of the aorta. The ductus arteriosus, which connects the trunk of the aorta with the left pulmonary artery during foetal life, should close as soon as the child's circulation is disconnected from that of the mother at birth. Sometimes its failure to close is explained by an absence of the usual fall in blood-pressure in the right side of the heart, and is due to imperfect opening up of the pulmonary artery. In a few cases no such factor is apparent, and the persistence of the ductus remains unexplained. The term coarctation denotes a narrowing of the aorta at that point where it is joined by the ductus arteriosus. The type of stenosis varies. In infants a diffuse narrowing of the "isthmus antæ," which extends from the origin of the left subclavian artery to the junction of the ductus arteriosus, is often associated with grave anomalies elsewhere; while in adults there is an abrupt constriction at the point of entrance of the ductus or a little below it. The necessary collateral circulation is established by opening up of the intercostal, phrenic, epigastric, and other vessels.

(d) Lastly, misplacements of the whole heart must be mentioned. The heart may lie in the right side of the chest (*dextrocardia*), a condition which may accompany partial or complete transposition of the viscera. This does not interfere with its functional efficiency. It may even lie on the wrong side of the diaphragm, in the abdominal cavity, without terminating life. The condition known as *ectopia cordis*, in which the anterior thoracic wall is imperfect and the heart lies on the outside of the body, is incompatible with life.

Morbid physiology.—Some malformations are so grave as to render the foetus incapable of maintaining a separate existence when parted from the mother; others exert no appreciable effect on the cardiac efficiency, the commonest of these being patency of the foramen ovale. The intermediate group is worthy of study; it consists of those malformations which constitute an appreciable hindrance to the circulation without going so far as to put separate existence beyond the bounds of possibility. These are the cases that come

under the notice of the clinician, and quite four-fifths are examples of pulmonary stenosis associated with patency of the auricular or ventricular septum or of the ductus arteriosus. The coexistence of one or more of these communications between the two sides of the heart with obstruction to the outflow of blood from the right ventricle is to be ascribed to the need which the latter experiences for providing itself with a safety-valve. The rise of intraventricular pressure caused by blockage of the pulmonary artery prevents the inter-ventricular channels from closing during embryonic life as they should. Consequently the pressure in the ventricles becomes equal, and the walls of the right ventricle become as thick as those of the left. The persistent overstrain which pulmonary stenosis inflicts on the right ventricle leads eventually to dilatation and failure, if life be not previously terminated by intercurrent infection or other causes.

Obstruction to the outflow from the right ventricle necessarily involves delay in the oxygenation of the blood. From this certain general effects arise, viz.: (1) deficient nutrition and stunted growth of the whole body; (2) increase of the number of red blood-corpuscles—an attempt on the part of the body to compensate for delay in oxygenation by providing an excess of oxygen carriers.

Symptomatology.—In two classes of congenital abnormality of the heart no symptoms arise—those in which the deformity is so grave that death ensues before the patient is old enough to manifest symptoms, and those in which the work of the heart is not interfered with appreciably by its error of development. As already pointed out, the residue consists largely of cases of pulmonic stenosis, usually associated with other deformities. In these the intensity of the symptoms varies with the degree of malformation present. *Dyspnoea* is constant, increasing with exertion and with age. *Cyanosis* is the most characteristic and striking symptom of congenital heart disease, as the term "morbus cæruleus" testifies. It is perhaps the best general measure of the degree to which the deformity is interfering with the cardiac functions, though one sometimes encounters bad cases of congenital heart disease in infancy with no cyanosis. The blue colour is diffused throughout the whole body, the mucous membranes and even the eye-grounds participating. It is usually increased by exertion, and also by cold. The connexion between cyanosis and pulmonary

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constriction is a very close one. Sometimes both the dyspnoea and the cyanosis which accompanies it are subject to paroxysmal exacerbations. This is especially characteristic of cases in which pulmonary stenosis is associated with patency of the interventricular septum at the undefended space—both ventricles opening, as explained above, into the aorta. These paroxysms may come on during exertion or while the child is being bathed, and give cause for much alarm to the parents. The red corpuscles are increased up to 10 millions per c.mm. in many of the patients with cyanosis.

The fingers are often clubbed, especially when there is much cyanosis (Plate 13). The same change is present as a rule in the toes, and even when the finger-ends are not actually thickened the Hippocratic finger-nail is noticed. These *peripheral changes* consist of a thickening of the connective tissues of the fingers and toes, bones and joints remaining unchanged. It is regarded as a result of deficient oxygenation. A similar explanation accounts for the *interference with the general growth* that is a common consequence of abnormalities which are severe without being fatal in the first year. Patients often feel the cold excessively. In a few advanced cases oedema of the legs appears. Cardiac pain is rare even in patients who survive childhood and reach the age at which the myocardium begins to deteriorate. Eclamptic and syncopal attacks occur, though rarely.

Apart from increased rate, the pulse does not usually depart from the normal. In many cases of congenital pulmonary stenosis the electrocardiogram is inverted, the ventricular complexes pointing down instead of up.

Physical signs.—The physical signs of congenital heart disease vary according to the precise form of deformity present. The signs most constantly encountered are those of *pulmonary stenosis*. A systolic murmur, usually harsh in character, is heard with maximum intensity at the pulmonary area, and is transmitted towards the left clavicle, and also to the back, being heard with maximum intensity in the left interscapular space. Rarely the co-existence of a ventriculo-septal defect or a patency of the ductus arteriosus may promote extension of the bruit into the vessels of the neck. In feeble infants this bruit may be difficult to hear, becoming more distinctly audible later if the patient survives. On the other hand, the bruit may be so intense as to be heard all over the chest.

Whenever the murmur is loud it is accom-

panied by a systolic thrill felt most distinctly at the pulmonary cartilage.

In most cases there are distinct evidences of enlargement of the right heart. The point of maximum impulse is not displaced, but the total area of pulsation is wider than normal, and in particular disproportionately powerful pulsation may be felt to the right of the sternum. In most cases it is possible to detect increase in the area of deep cardiac dullness to the right of the sternum. Examination with the X-rays is also of great value in demonstrating increase in the size of the right heart. Both by this means and by percussion the shape of the heart appears to be abnormally globular. Since it is usual to find pulmonary stenosis associated with other malformations, these signs may be combined with those which indicate the presence of other deformities. Defect of the auricular septum, more often than not, causes no physical signs. Happily its presence is of little prognostic importance, whether it is associated with pulmonary stenosis or occurs alone.

Patency in the interventricular septum announces itself much more unmistakably when it occurs alone than when it is associated with pulmonary stenosis, because the bruit that is its chief characteristic blends with that of the latter lesion when they coincide. In its most typical form it is a loud systolic murmur heard with maximum intensity over or immediately to the left of the mid-sternum, at the level of the fourth interspace; it is transmitted in no special direction, but is audible all over the chest. There is often a corresponding thrill.

The signs of *patency of the ductus arteriosus* are rather more distinctive. Along the left border of the upper sternum a strip of dullness is often found; over the left upper front a bruit is heard, the main characteristic of which is that it is continued from systole into diastole. It is often very loud, and gives to the uninitiated quite a disproportionate sense of its importance. It is sometimes accompanied by a thrill, and this, together with the bruit, may be transmitted into the neck as well as into the interscapular space. With the fluorescent screen the cardiac shadow is seen to extend abnormally in an upward direction; this is ascribed sometimes to dilatation of the pulmonary artery, sometimes to lifting up of the aortic arch by the dilated ductus arteriosus. These signs, like those of patent interventricular septum, may occur alone or in combination with those of pulmonary stenosis.

HEART, CONGENITAL DISEASE OF

The majority of the other gross deformities of the heart are of little clinical importance, so rarely do their victims survive birth. *Coculation of the aorta*, however, deserves mention. A loud systolic murmur is heard all over the chest, and particularly in the left interscapular space, and—what is most characteristic—collateral circulations between the upper and lower parts of the body develop in the epigastric and other superficial arteries; the carotids also become disproportionately large. The last sign is observed only in adults.

Course and terminations.—Many of those who survive birth die in early infancy from marasmus or from intercurrent pulmonary infections. To the latter, and especially to tuberculosis, the subject of congenital heart disease is prone at all ages. Tuberculosis is the cause of death in a large proportion of cases. Malignant endocarditis accounts for death in many cases, circulating micro-organisms apparently obtaining a hold on the malformed heart—for instance, on the edges of a septal foramen—with much greater ease than on a normal heart. Those who escape these dangers may die from cardiac failure, either sudden and syncopal, or gradual and associated with cedema and pulmonary congestion, just as in cases of longstanding acquired heart disease. It is rather surprising, however, how seldom this gradual cardiac breakdown terminates congenital cases.

Diagnosis. (a) *From conditions simulating heart disease.*—In many feeble infants very distinct bruits are heard over the heart. Practically always they are systolic, and generally they are equally audible at all areas. This latter fact, together with the absence of symptoms and of right-heart enlargement, should serve to exclude the presence of an organic defect. A somewhat similar fallacy is that provided by the venous murmurs often heard in young children, especially if they are anæmic, on either side of the manubrium sterni. Since these are usually continuous from systole into diastole, they may be mistaken for the bruit of patent ductus arteriosus. Their variability with posture, and the absence of the other signs of congenital malformation, are the chief points of discrimination. Of the other varieties of exocardial bruit, that which most closely simulates the bruit of pulmonary stenosis is the systolic murmur heard in children at the pulmonary area in association with atelectasis of the left upper lobe. It is the more likely to cause confusion because of its association

with atelectasis, and consequently with distinct cyanosis. The characteristic features of exocardial bruits (see HEART, FUNCTIONAL MURMURS OF) serve to distinguish them from those of organic heart disease.

(b) *From acquired heart disease.*—Difficulty seldom arises in actual practice. The principal points of distinction are the history and the distribution of the bruits. Occasionally a case of patent interventricular septum is mistakenly regarded as one of rheumatic mitral disease, but the increase in the size of the heart is to the left in the latter and to the right in the former, and in mitral disease the bruit is transmitted pre-eminently to the left, while in the congenital disease its maximum intensity is central.

(c) *Between the various forms of defect.*—Great accuracy in diagnosis is not possible. The principal points to remember are that pulmonary stenosis is present in four-fifths of the cases of congenital heart disease diagnosed during life, and that it is usually accompanied by one or other of the lesions whose distinctive signs are enumerated above. The value of the X-rays should not be forgotten.

Prognosis.—Cure of congenital heart lesions is, of course, out of the question. Nevertheless the expectations as to the duration of life and extent of activity have to be assessed. This may be attained by discovering the nature of the malformation and measuring the extent of interference with the cardiac functions. Pulmonary stenosis is not incompatible with the patient's reaching adult life. The more pronounced the evidences of imperfect aeration of the blood (dyspnoea, cyanosis, polycythæmia, clubbing of the fingers) the worse the outlook. Uncomplicated patencies of the septa, whether of the auricle or ventricle, are compatible with the attainment of middle life and the exercise of fair activity; and the same is true of patency of the ductus arteriosus. The average age at death in pulmonary stenosis is about 9, and in pulmonary atresia 3.

Of combined lesions, the association of pulmonary stenosis with defective auricular septum spares life longer than when the former lesion coincides with a patency of the interventricular septum. Ectopia cordis is fatal within a day or two of birth if the heart lie exposed on the anterior wall of the chest; but transposition of the heart has no influence whatever on the expectation of life. Apart from the evidences of defective aeration enumerated above, serious signs are those which

HEART, DILATATION OF

indicate pulmonary infection (tuberculosis or pneumonia), malignant endocarditis, or failure of compensation. The sudden and alarming attacks of cyanosis and dyspnoea that may occur in cases of pulmonary stenosis with patent interventricular septum are not so dangerous as they appear.

Treatment.—Very little can be done for the unfortunate victim of cardiac malformation. During infancy and early childhood he should be protected from cold and wet (special attention being paid to the need for woollen underclothing), and as far as possible from the risk of infection with measles or whooping-cough. His diet should include a generous proportion of fat. As he grows up, lessons, games, and eventually his occupation, must be regulated by a knowledge of what his limited cardiac abilities will allow him to do without getting out of breath. It must never be forgotten that he is abnormally susceptible to tuberculous infection, and this risk must be considered in every important decision as to occupation, place of habitation, and general environment. Attacks of bronchitis are treated along ordinary lines with stricter care than is usually needful. In the later stages, if pain arises, it may be controlled by nitrites. Signs of cardiac failure should be combated by rest in bed and the other measures which are generally useful in such circumstances. One word of caution is necessary, especially in connexion with cases (for instance, of patent ductus arteriosus) where the outlook is not so gloomy: the patient should never be discouraged by too much insistence on the presence of cardiac disease. I have seen patients with a relatively harmless malformation worried into a condition of nervous invalidism by a continual harping on the presence of the abnormality.

CAREY COOMBS.

HEART, DEGENERATIONS OF (*see* MYOCARDIAL DEGENERATION, PROGRESSIVE).

HEART, DILATATION OF.—This is not a disease but a symptom. It occurs as a result of disease attacking the myocardium. The healthy heart does not dilate. An increase in the transverse diameter of the cardiac dullness, and leftward enlargement of the area of cardiac pulsation, are the usual signs of dilatation. Orthodiagraphy gives more precise information, but this need seldom be resorted to. The chief importance of discovering the presence of dilatation is that it should provoke the observer to search for a cause.

CAREY COOMBS.

HEART, FUNCTIONAL MURMURS OF

HEART, FUNCTIONAL MURMURS OF.—Functional murmurs fall into two groups: (1) those in which the bruit is produced within the heart or great vessels, but without being indicative of disease thereof, and (2) those in which the origin of the murmur is exocardial. The importance of functional murmurs lies almost exclusively in their imitation of those indicative of organic heart disease, and in the mistakes in diagnosis to which this may lead.

1. Functional murmurs produced within the heart or great vessels.—The best-known class are the so-called "hæmic" bruits. The name is due to the idea that a poor quality of the blood is responsible for their production. It is probable, however, that anæmia causes such bruits only indirectly, through its influence on certain of the cardiac functions. Bruits of this class are encountered not only in every kind of profound anæmia, but also in acute infections of every description, and particularly in children. Any infective process which raises the temperature to 102° F. or so seems capable of causing these murmurs in children. Their most characteristic features are—(a) that they are always systolic; (b) that they are nearly always louder at the base of the heart than at its apex, the third left costal cartilage marking their point of maximum intensity; (c) that their area of transmission is small; (d) that they occupy a relatively short part of systole; and (e) that they are very rarely harsh in character. The mechanism of their production is obscure. As a working hypothesis we may accept the view which ascribes the apical systolic bruits to relative and temporary incompetence of the mitral valve, caused by depressed tonicity of the anæmic or poisoned cardiac muscle. According to this view the basic bruits are similarly produced by loss of tone and consequent yielding of the walls of the great vessels as they leave the heart; the result being that blood flows through the fibrous, unyielding cardio-arterial rings into the widened arterial channels, setting up vibrations which are audible as a systolic murmur. This theory is satisfactory in one respect, for it explains the difficulty, experienced clinically, of drawing a hard-and-fast line between the systolic bruit of mitral regurgitation and the functional apical murmur of the type described. In a given case, the more nearly a murmur conforms to the features mentioned above, the more justification is there for regarding it as functional and transitory.

HEART, FUNCTIONAL MURMURS OF

Certain other bruits heard at the base of the heart and in the neck are produced within the great vessels and imitate those of organic disease. (a) Such, in anæmic girls, is the "bruit de diable," a systolic hum heard loudest over the great veins at the root of the neck. If confined to this area it is not likely to be mistaken for a murmur caused by organic heart disease, but in a few instances it is also heard over the manubrium. (b) In many children, both normal and abnormal, a bruit can be heard over the sterno-clavicular joints; it begins in the middle of systole, persists into diastole, and increases in intensity when the head is bent back. If this extend downwards it may simulate the continuous murmur of a patent ductus arteriosus or the diastolic bruit of aortic regurgitation. (c) Below the clavicles a systolic bruit may be heard in quite healthy persons, especially when the arms are abducted from the trunk. This bruit, which originates in the subclavian arteries, has no connexion whatever with heart disease, but it may sometimes simulate aortic or pulmonary systolic murmurs and thus lead the unwary into error.

2. Murmurs which arise outside the heart but simulate those of cardiac disease.—In the tuberculous, as well as in healthy but excited persons (e.g. during an insurance examination), a systolic bruit is often heard at the apex of the heart, along the cardio-pulmonary borders and even at the inferior angle of the left scapula. This is the commonest type of cardio-pulmonary or cardio-respiratory murmur. It is probably caused by friction between the apposed pleural surfaces under the influence of the heart-beat, and is not due to cardiac disease. Its characteristic features are—(a) that it is louder during inspiration than expiration; (b) that it is louder when the patient is standing up; (c) that it begins rather late in systole; (d) that it has a short, jet-like character. It is often mistaken for the systolic murmur of mitral regurgitation, but its respiratory and postural variations are so characteristic that they serve to make the diagnosis easy in most cases.

At the pulmonary area in children, with collapse of the anterior border of the left lung, quite loud murmurs may be heard in systole. As a rule these are strictly localized, and occur late in systole. The presence of signs of pulmonary collapse and the absence of collateral evidence of congenital heart disease usually enable the observer to exclude the

HEART, HYPERTROPHY OF

latter condition. In healthy infants and children a similar murmur is often heard; no notice need be taken of it unless it is accompanied by other signs of congenital heart disease.

To sum up: No murmur should be accepted as evidence of organic heart disease until it has been carefully scrutinized from the following points of view, viz. its variability with respiration or posture, its relation to the first and second sounds of the heart, its localization in relation to the areas to which organic cardiac murmurs are usually limited, and the presence or absence of other symptoms or signs of cardiac disease.

CAREY COOMBS.

HEART, HYPERTROPHY OF.—Hypertrophy of cardiac muscle is caused by persistent increase in the work demanded from the ventricles, as in aortic incompetence, where a larger bulk of blood has to be thrown by each systole, and in chronic renal disease with high blood-pressure. The impulse becomes powerful and heaving in character, and widened in area. Its maximum point is displaced downwards and outwards in most cases because it is usually the left ventricle that is concerned. If it is the right side that is affected, powerful pulsation will be visible in the epigastrium. In doubtful cases of right ventricular hypertrophy, skiagraphy and electrocardiography may assist in arriving at a confident decision. The discovery of the signs of hypertrophy should prompt a search for the cause.

Hypertrophy is a compensatory physiological effect and beneficent in its action, preventing symptoms rather than causing them, and in itself needs no treatment. An exception should perhaps be made to cover the case of men who do exceptionally hard physical work, and whose heart is compensated for their active hours. During rest such men often complain of forcible thumping in the chest, headache, sleeplessness, throbbing in the head and ears. These symptoms have been ascribed to the hypertrophy itself—an hypertrophy which is proportionately too great for the inactive part of the twenty-four hours.

CAREY COOMBS.

HEART, IRRITABLE (Disordered Action of the Heart). **Etiology.**—This symptom-complex arises from many causes. The strongest predisposing factor is the high-strung anxious temperament. Possibly puberty, and probably the climacteric, are susceptible ages. Of the exciting causes, the most important are

HEART, IRRITABLE

emotional stress of the "inner conflict" type, and various infections. It is the coincidence of these factors that increased the incidence of the syndrome during war years.

Pathology.—All are now agreed that this does not lie in any myocardial lesion, but in some disturbance of the cardiac innervation; but opinions differ as to the actual point of attack.

Symptoms.—The patient complains of cardiac discomfort of the "palpitations" type, excited by emotion or moderate exertion; of dyspnoea, præcordial pain, giddiness, and even fainting. The pulse is fast, either constantly or on slight provocation by emotion, exertion, or change of posture. Periods of abrupt retardation occur in some cases, and may be accompanied by premature beats. It is in the periods of slowing that the faints are apt to occur. The systolic pressure tends to be high and the diastolic pressure low. There may or may not be murmurs over the heart. Even when these, together with cardiac enlargement, prove the existence of an organic lesion, the relation between this and the tachycardia is one of coincidence only. In the typical "irritable heart" case there are no signs of organic heart disease. The expression is often anxious, the tendon-jerks are increased, nutrition is poor, and sleep disturbed.

Diagnosis.—Such conditions as *phthisis* and *Graves's disease* have to be remembered. As "irritable heart" is not a disease but a symptom-complex having many causes, these causes have to be identified. Even when an infection can be blamed, there is usually some emotional stress in the background.

Prognosis.—If the cause can be removed, the patient will get well. Recovery is protracted, especially if the symptoms have existed for a long time. The condition is never fatal, but its coincidence with grave cardiac disease increases the seriousness of the latter by adding to the work of the heart.

Treatment.—Tell the patient his heart is not diseased, and explain simply the origin of his troubles. Attack the cause: if an infection, by removing it or raising resistance; if psychical, by the same means—adjusting the situation or educating the patient to bear his trouble calmly. Systematic physical exercise is good, as also is mental occupation. Patients ought not to be put in bed, except for a few days' rest if there is loss of flesh and profound debility. Drugs are not indicated, except as a part of an attack upon the cause, e.g. quinine in malarial cases.

CAREY COOMBS.

HEART, PALPITATION OF

HEART, PALPITATION OF.—The term "palpitation" is used to signify that a patient is conscious of the beating of his heart. Associated with palpitation there is usually, though not invariably, an increase in the rate. The amount of distress varies considerably; sometimes it is great, and may cause apprehension and restlessness; not infrequently the patient is conscious merely of the beating of his heart and is in no way perturbed. During the attack the skin may be flushed or pale, and is often unduly moist. Sometimes the whole præcordium shakes with the tumult, or in contrast there may be no apparent variation from the normal. If the hand be placed over the cardiac thrust and firm, gentle pressure applied, a sense of comfort and relief is experienced. At the same time it will be noted that the normal steady push of the apex beat is altered to a short, sharp, throbbing slap, and this accentuated staccato beat with its suggestion of overaction seems to be out of all proportion to the pulse felt at the wrist, which is often weak and of poor tension.

A sharp attack of palpitation often gives rise to pain and tenderness over the heart, and, even after the paroxysm has passed, pressure over this area may be intolerable.

The onset may be gradual or sudden; a mental shock, the receipt of ill news, the slamming of a door, or some similar stimulus may send the heart racing.

The presence of palpitation is often nothing more than an additional evidence of an extremely labile vaso-motor system, and may be quite independent of any organic disease. It is an outstanding feature in the so-called D.A.H. of soldiers. It is more common in women than in men, and in the highly-strung than in the phlegmatic. During the menopause it frequently accompanies "flushing" and similar manifestations.

In patients with high blood-pressure it is sometimes a very distressing symptom. Palpitation, like other manifestations of functional disturbance, may be the dominant symptom in cases of organic disease, and this is especially the case in early mitral stenosis, where palpitation and rapid heart-action are common.

In frank organic disease of the heart, with a diminished reserve power, effort induces increased frequency of the beat, and under these conditions the patient may be conscious of his heart-action. Palpitation is then definitely associated with effort.

HEART, PALPITATION OF

In the neurotic or toxic forms, palpitation, though sometimes induced by effort, more often comes on independently, and an attack is more likely to be caused by emotional disturbance. Again, it frequently starts after the patient has retired to bed, causing restlessness, discomfort, and insomnia.

One form of palpitation is due to the occurrence of extrasystoles, and these, especially if frequent, may cause considerable discomfort. In some subjects the peculiar sensation induced by premature beats is distressing in the extreme.

Palpitation is also an important symptom in many cases of tachycardia with a pathological rhythm, such as paroxysmal tachycardia, auricular flutter, and auricular fibrillation.

Alcohol, tobacco, tea, and coffee are the chief toxic agents responsible, and in many highly-strung individuals a strong cup of tea or coffee late in the evening is quite sufficient to cause palpitation and insomnia.

If the heart be auscultated during the height of the attack, a short whiffy systolic bruit is often audible; this is not permanent and is inaudible when the attack has subsided.

The **prognosis** in any particular case must be arrived at from a consideration of all the factors. Palpitation in itself is not ominous; when it develops late in life it has greater significance.

Treatment necessitates a wide survey, and every means must be used to improve the general condition, such as graduated exercises and games in the open air, a rapid cold sponge-down in the morning, careful regulation of the bowels, and a reasonable and appropriate diet. The possibility of a toxic origin must be considered, and the amount of alcohol, tea, coffee, and tobacco carefully restricted. Any factor undermining the stability of the nervous system, such as insomnia, overwork, mental strain, anxiety, or worry, should be removed or mitigated as far as possible.

Anæmia requires iron and arsenic, and where there is organic disease of the heart or a pathological rhythm, appropriate treatment must be ordered.

During an attack the patient should rest. If the palpitation persists, cold applications to the præcordium are of service. A mustard leaf or a belladonna plaster sometimes gives relief.

Sips of very hot water or a draught of menthol in sal volatile and spirit of chloroform in water are useful. If there is a history of

HEART, SYPHILITIC DISEASE OF

insomnia, anxiety, or worries, especially if the patient is at the menopause, a course of bromide of ammonium and arsenic should certainly be prescribed.

JOHN HAY.

HEART, SYPHILITIC DISEASE OF.—

In acquired syphilis, infection of the myocardium and of other parts of the heart occurs as early as during the late secondary stage. By the time the tertiary stage is reached the lesions and their effects have attained to a more pronounced degree. They coincide not infrequently with late lesions of the central nervous system (tabes, general paralysis, and ophthalmoplegia interna). The heart is more often attacked in males than in females. In congenital syphilis also, infection of the myocardium (Warthin) and of the aorta is encountered, but this is a pathological rather than a clinical entity even more than in the case of the acquired infection. Some claim that congenital malformations are more frequent in the children of syphilitic parents. More than half of all cases of aortic regurgitation and of diffuse aortic dilatation are syphilitic in origin. It is difficult to say what fraction of the primary myocardial degenerations of middle life are syphilitic, but it is certainly large.

Pathology.—The lesions found post mortem, excluding those of the aorta and its valves, which are considered elsewhere, are as follows:—

Coronary arteries with myocardium.—Obstructive inflammation of the coronary vessels is a constant feature of the disease. If a large vessel is damaged, or if thrombosis occurs as a result of the arteritis, gross changes follow (see CORONARY ARTERIES, DISEASES OF); if finer twigs are implicated, the dystrophic lesions of the myocardium which result are less obvious but none the less real. Scattered areas of fibrosis constitute the commonest type of naked-eye change; microscopically these are associated with degenerative changes in the neighbouring muscle-fibres. Much more rarely, conspicuous gummata are present. Spirochætes have been found in these foci in the earlier inflammatory stages, and also in the inflammatory and necrotic areas which may occur in the myocardium in congenital syphilis.

Pericardium.—Adhesive mediastino-pericarditis is rare. Patches of the pericardial thickening may be associated with focal lesions of the myocardium.

These changes may occur not only singly but

HEART, SYPHILITIC DISEASE OF

also in various combinations; for example, aortitis is almost invariably associated with some degree of myocardial injury.

Symptomatology.—The groundwork is that of myocardial disease, but to this may be added the symptomatology of aortic valvular disease or of aneurysm, more rarely of sudden coronary obstruction, and these syndromes, though subsidiary in origin, may overshadow clinically the symptoms due to myocardial disease. The chief of these is pain, which may be anginal or subanginal in degree. The association of pain of cardiac type with little or no physical evidence of myocardial disease is very characteristic of myocardial syphilis, and the patients may die suddenly without having given much hint of anything wrong. Dyspnoea on exertion is another common symptom, but cases do not often linger on into the chronic bedridden condition that so often forms the last phase of other myocardial degenerations. Of the various evidences of focal disease of the myocardium, heart-block is deserving of particular mention. It is, of course, manifest in a small proportion of cases only, but when it does occur it affords undeniable evidence of a focal myocarditis.

Physical signs.—From this point of view the cases may be divided into four groups: (1) Some manifest symptoms without any definite signs of disease. (2) In this group are included those patients in whom the only physical signs are more or less obscure evidences of myocardial disease—signs of dilatation and hypertrophy of the left ventricle predominating. Signs of aneurysm of the heart and of sudden rupture into the pericardium should also be mentioned as very rare features of such cases. (3) In this, a large group, the predominant signs are those of aortic valvular disease. (4) Finally, another large group consists of those cases in which the predominant signs are those of aortitis. These are—(a) signs indicative of diffuse dilatation: dullness in a small area on either side of the manubrium, limited pulsation in the aortic area, accentuation of the aortic second sound, and general increase in the skiagraphic shadow of the aorta; (b) rarely, pressure signs such as inequality of the pupils or distension of the upper intercostal veins; (c) signs of implication of the mouths of branch arteries in the aortic patches, the chief being diminution or even obliteration of one of the radial pulses. Various combinations of these groups of physical signs may occur. Thus, evidences of myocardial

disease usually accompany those of syphilitic aortitis; and it must not be forgotten that the vast majority of aortic aneurysms are founded upon a basis of syphilitic aortitis, so that signs of aneurysm are often associated with those of other forms of cardiac syphilis.

Diagnosis.—The first task is the discovery of the presence of organic heart disease. The chief point is to remember that symptoms such as angina or persistent dyspnoea on exertion should be treated with due respect even in the absence of physical signs of cardiac disease, especially if there be evidence of syphilitic infection. Secondly, the presence of cardiac disease being established, is it due to syphilis? Here three things should be remembered: (1) the paramount influence of syphilis in provoking symptoms without signs of myocardial disease and in causing lesions of the aorta and its valves; (2) the value of the Wassermann test; (3) the presence of other evidences of syphilitic infection, such as tabes, gummata of bone, etc.

Prognosis.—Though cardiac syphilis is to some extent amenable to treatment, the end-results are disappointing. The average duration of life from the onset of symptoms is not more than six years, even if the cases of aortic aneurysm coinciding with true cardiac syphilis be omitted. Sudden death is fairly common both in cases with signs of aortic regurgitation and in those which present only indistinct evidence of myocardial disease.

Treatment.—The causal infection must be vigorously attacked. To this end thorough mercurialization is necessary, either by inunction or by intramuscular injection or orally. Potassium iodide should also be pushed, intervals being allowed according to the progress of the case. The propriety of using salvarsan in such cases is still under discussion. Some hold that it ought to be used in all cases of cardiac syphilis, others that the presence of distinct cardiac disease is an absolute contraindication. My own view is that when cardiac syphilis has reached the point at which it can with certainty be diagnosed, it has already advanced beyond the stage of curability, so that the salvarsan treatment exposes the patient to a certain risk for the sake of a doubtful benefit. If arsenobenzol is given, it should be in the form of neosalvarsan; the first dose should not exceed 0.15 grm., and if the dosage is increased from dose to dose an interval of a week should be allowed between one injection and the next.

HEART-BEAT, ABNORMALITIES OF

Apart from this attack on the causal infection, its results must be dealt with as they arise. Pain is relieved by the nitrites. Rest in bed should be insisted upon whenever the symptoms of myocardial insufficiency seem to be gaining ground. As a general rule, the extent of liberty allowed to the patient should be conditioned rather by symptoms than by physical signs, but any person who is a proved subject of cardiac syphilis is unfit for sudden strain, emotional stress, or strenuous occupation.

CAREY COOMBS.

HEART, VALVULAR DISEASE OF, ACUTE (see ENDOCARDITIS).

HEART, VALVULAR DISEASE OF, CHRONIC (see VALVULAR DISEASE, CHRONIC).

HEART-BEAT, ABNORMALITIES OF.

—The great service of the polygraph and electrocardiograph has been to permit the separation of abnormalities of the heart-beat into their several varieties. The prognostic significance of the varieties thus identified has been determined largely through the labours of Sir James Mackenzie, who for years watched patients presenting them until their significance became manifest. To recognize a pulse as irregular is worthless unless we distinguish the particular kind of irregularity. This is now possible in most cases without the use of graphic records. But the polygraph and electrocardiograph were the means of isolating them, and still remain the most accurate means of diagnosis.

THE POLYGRAPH

The clinical polygraph is a modern development of the old sphygmograph, which registered on smoked paper the radial pulse alone. The improvement lies in (1) the use of two tambours for recording simultaneously both the jugular and the radial pulsation, and (2) the substitution of ink and a long paper roll for the short strip of smoked paper. The jugular curve furnishes information on auricular activity, about which ordinary clinical examination and the sphygmograph reveal nothing.

The ink polygraph devised by Sir James Mackenzie consists of a body bearing the recording parts of the apparatus and containing two clockwork mechanisms, one to pass the paper at a chosen rate under the pens, the other to drive a time-marker registering $\frac{1}{2}$ seconds in ink. At the back is the roll of re-

cording paper; in front is attached a firm rod carrying the two small tambours with their respective long pens which write upon the paper as it passes over the body of the machine. The recording tambour adjacent to the supporting rod is connected by 2 ft. of light rubber tubing with a large radial tambour which rests upon the wrist-splint and so receives the pulsation of the radial artery. The other recording tambour is similarly connected with an open metal receiver which, applied to the neck, receives the jugulo-carotid pulsation.

In use, the polygraph is assembled on a firm table or locker placed to the right of the head of the bed. The patient should lie at ease with the head as low as possible, yet supported so as to relax the sterno-mastoids. When the apparatus is in working order the observer sits down, marks the line of the radial artery at the right wrist, and applies the wrist-splint. The spring should lie along the artery so that its projecting button acquires the pulsation. If need be, it should be applied again and again until the spring shows an ample excursion. The lower strap should be fixed first and rather tightly, then the upper strap loosely. The difficulty is to leave the spring button exactly over the pulse when the strap has been buckled, but the lateral displacement inseparable from this will be allowed for unconsciously with practice. The spring tension regulator will sometimes amplify the pulsation thus obtained. The wrist may lie upon a small folded towel so that the hand hangs over it, preferably with ulnar deviation, for this throws the radial artery into relief. The wrist-tambour is elevated on its rod, affixed to the wrist-splint, and allowed to descend and rest upon the spring. The corresponding pen should begin to move freely with the pulse. The neck-receiver is then applied to the skin just above and not too far external to the right sterno-clavicular joint. If the rim of the receiver is completely in contact with the skin, the second pen now begins to move with the venous pulse. The moving pens, charged with ink, are made to touch the paper lightly, and the clockwork is released by the lever. After the requisite tracing is obtained, ordinates are drawn by moving each pen up and down on the paper, and the polygram is complete. If desired, the neck-receiver may be used to obtain a record of apical, epigastric, or aneurysmal pulsation. Respiration is recorded by connecting in its stead a small rubber bag inserted under the patient's clothing. If ever the excursion of a

HEART-BEAT, ABNORMALITIES OF

pen is feeble, though the visible pulsation is great, a leak in the air system is probable. The rubber tambours deteriorate, and may be replaced by toy balloon rubber. If the pens lie badly on the paper, it may be that the rod supporting the tambours has not been pushed well into the slot. Red ink flows more freely than black. The pens should be gently wiped dry with gauze after use, and occasionally cleansed in spirit or hot water.

form, of equal height, of normal rate, and equidistant. The rate is ascertained by measuring six seconds ($30 \times \frac{1}{5}$ sec.) with dividers and transferring these to the radial curve. The number of radial beats enclosed, along with the odd fraction as a decimal (e.g. 7.5), give the rate during six seconds, and by omitting the decimal point the rate per minute (e.g. 75).

(2) **The jugular curve** (Fig. 32).—Each heart-beat or cycle is represented on a jugular tracing

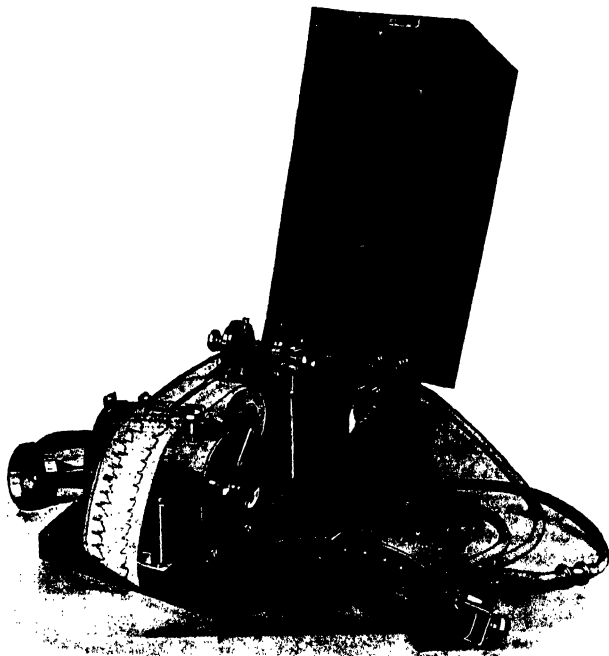


Fig.—31.—Sir Thomas Lewis's modification of Mackenzie's original ink polygraph.

A modified form of the original ink polygraph has been recently introduced by Sir Thomas Lewis (Fig. 31). It is used as described above, but a glycerin pelotte is supplied in place of the ordinary wrist attachment.

The normal polygram. (1) **The radial curve.**—The form of isolated radial beats is not now considered of much diagnostic value; stress is rather laid upon the time-relation of one beat to another—the rhythm. In a normal pulse-curve the beats are of the same

by three principal waves, *a*, *c*, and *v*: *a* is due to auricular systole, *c* is the carotid wave due to ventricular systole, *v* is the wave of venous stasis while the ventricle continues in contraction.

To analyse a jugular curve, *c* must first be identified by measurement. Dividers are applied to the subjacent radial curve, one point to the ordinate at the end, the other point to the beginning of a chosen radial beat. The dividers are then separated $\frac{1}{5}$ sec. wider, for

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this is the difference in time between carotid and radial pulsation. If now they are transferred to the jugular tracing with one point on the ordinate, the other point on the left will indicate the beginning of the *c* wave. The wave seen immediately preceding *c* is

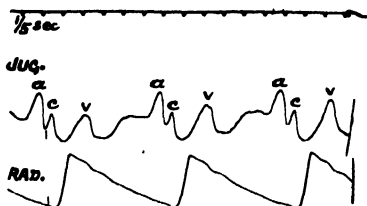


Fig. 32.—Normal polygram, showing three heart cycles. *a* = auricular systole; *c* = carotid wave, the beginning of ventricular systole; and *v* = venous stasis wave at the end of ventricular contraction. The radial beat occurs $\frac{1}{10}$ sec. after the carotid wave.

presumed to be *a*, and the normal interval between them (*a-c* interval) is $\frac{1}{2}$ sec. or less.

The *v* wave, of less importance in analysis, is recognized by its fixed relation following *c*, and by its correspondence with the dicrotic notch of the radial beat. The period *c-v* may be regarded as the period of ventricular systole.

THE ELECTROCARDIOGRAPH

All muscular contraction is accompanied by changes in electric potential, the active part becoming negative to the passive. When the heart begins to contract, the part first in contraction becomes electrically negative to the rest of the heart. Then as the wave of contraction passes down the heart a wave of negativity passes with it. The different phases of the heart's beat are accompanied by characteristic variations in the faint current produced, and these are the variations recorded by an electrocardiograph. The current is drawn from the body through electrodes, one applied to the arm (for the base of the heart) and the other to the leg (for the apex). The circuit is completed by wires leading away from these electrodes to a fine silvered glass thread which is suspended within the field of a large magnet in the midst of the apparatus. When the heart beats and the varying current passes, it deflects correspondingly this sensitive thread. The magnified shadow of the moving thread is projected, by means of an arc lamp and a microscope, upon the slit of a special camera.

A falling photograph plate receives the shadow deflections of the thread and converts them into a continuous record—an electrocardiogram.

The normal electrocardiogram (Fig. 33).—There are three main waves to each cardiac cycle, (1) *P* = auricular systole, (2) *R* = beginning of ventricular systole, (3) *T* = end of ventricular systole. For practical purposes *P* is the electrical expression of auricular systole, as *a* is its pulse expression in the jugular curve of a polygram. Similarly, *R* represents electrically the onset of ventricular systole and inasmuch corresponds with *c*, the carotid wave of a polygram. The final deflection, *T*, is scarcely comparable with *v* of the venous pulse, except that both occur towards the end of ventricular systole.

The commonest variations in disease include absence of the *P* wave in auricular fibrillation, excessive rate of *P* (e.g. 300 a minute) in auricular flutter, and increase in the *P-R*, or auriculo-ventricular interval, in heart-block.

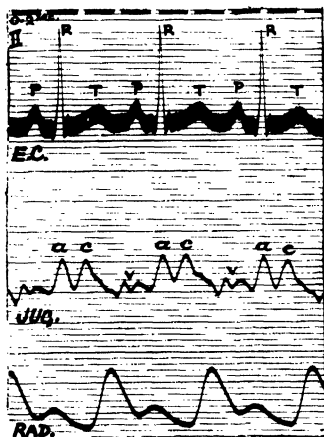


Fig. 33.—Normal electrocardiogram, with simultaneous jugular and radial curves. *P* and *a* represent auricular systole; *R*, *c*, and the radial beat indicate the beginning of ventricular systole.

The height of the respective waves is not a guide to the force of the beat; electrocardiograms give information chiefly on rate and rhythm. In addition, a comparison of the records taken from the three standard "leads"

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(R. arm and L. arm, R. arm and L. leg, L. arm and L. leg) furnishes evidence on the preponderance of left over right or right over left ventricular muscle, and therefore on hypertrophy.

SINUS ARRHYTHMIA

The sino-auricular node or "sinus," situated at the junction of the superior vena cava with the right auricle, is the birthplace of the normal impulse which determines each heart-beat. This node, the pacemaker of the heart, is supplied by the vagus, which thereby controls the rate of the heart. Acceleration of the pace may be due to minimal, retardation to maximal vagus action. There is, for example, a simple tachycardia when the vagal action is eliminated as by atropine, and a

under digitalis, a phasic variation of rate may occur without such direct relation to breathing.

Sinus arrhythmia is unfortunately still often confounded with arrhythmia of serious significance. Many children have been consigned to bed for months on account of this harmless irregularity. Its recognition is simple because it is so clearly related to respiration. If there is a doubt, a combined radial and respiratory polygram will decide. Treatment is superfluous.

PREMATURE CONTRACTIONS

This name is preferable to *extrasystole* because the abnormal beat is only a premature, not an additional or extra, contraction. A premature contraction is the result of a stimulus originating at some abnormal site in the myo-

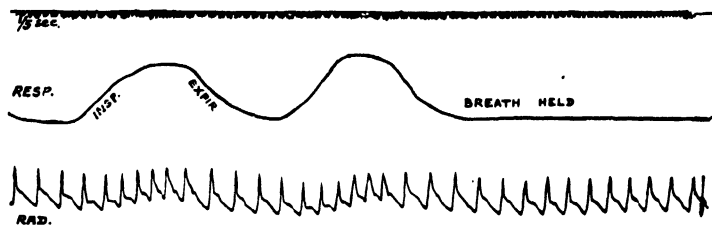


Fig. 34.—Sinus arrhythmia. Respiratory and radial polygram showing the quickening of the pulse during respiration, and the slowing during expiration. When the breath is held, this physiological arrhythmia disappears.

simple bradycardia when the vagus is over-acting.

The proximity of the respiratory to the vagal centre lends itself to the transference of slight variations in vagal tone with each respiration. This vagal effect is diminished during inspiration leading to a momentary quickening of the heart, and increased during expiration leading to temporary slowing in this phase. This normal respiratory variation in the heart-rate is often recognizable at the pulse as an irregularity—*sinus arrhythmia*.

It is important at once to realize that this irregularity is physiological and has no sinister significance whatever. It is most noticeable when the pulse is slow, and in the young, whence the term *juvenile arrhythmia*; yet in a lesser degree it may be present with higher rates and at any age. It consists of a quickening of the pulse with inspiration and a slowing with expiration; it is exaggerated by deep breathing and disappears when the subject holds his breath (Fig. 34). In rare cases, and

cardium, and it occurs earlier than the normal beat would be expected.

Etiology.—Premature contractions are so common in adults of all ages, especially in the elderly, that it is difficult to ascribe them to any particular pathological condition in the heart; usually there is none. Arising during an acute infection, e.g. rheumatic fever, they may perhaps depend on acute myocarditis, and sometimes they are provoked by narrowing of a coronary artery or its branches.

Symptoms.—Many people are known to have an intermittent pulse due to this irregularity for years, or even a lifetime, without experiencing any symptoms, and without showing any signs of disease. Others recognize the occurrence of premature contractions by a peculiar sensation over the heart, but take no further notice of them. A few patients are really troubled either because the premature contractions are numerous and persistent or because their nervous system is more impressionable. They complain of palpitation and

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describe their sensation as a sinking feeling at the heart or stomach, often with a momentary sense of choking at the throat. They may say that the heart seems to turn over, or that it stops (the premature beat unnoticed) and then gives a thump (the succeeding large beat). Though such sensations may be felt by anyone subject to premature contractions, it is chiefly among neurasthenics that they are magnified into a serious complaint. They are usually most evident when the pulse is infre-

ture contractions are numerous, the resulting irregularity may simulate that of auricular fibrillation, but a few arm exercises will increase the heart-rate and dissipate premature contractions, while rendering the grosser irregularity of fibrillation more obvious.

The diagnosis can be established by a polygraphic or electrocardiographic record, which will also reveal the site of origin of the errant beat. (1) Premature *auricular* contractions take origin at some point in the auricular wall.

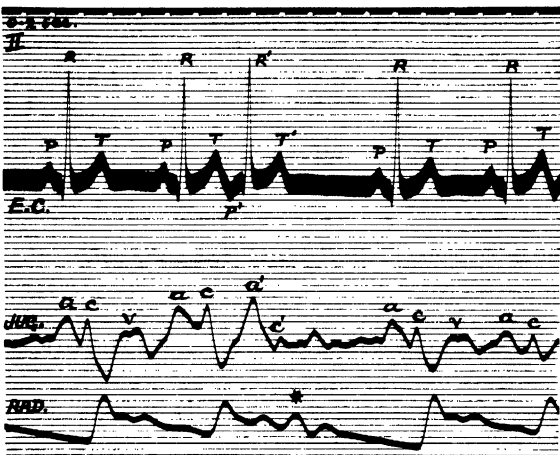


Fig. 35.—Premature auricular contraction. Simultaneous record of electrocardiographic, jugular, and radial tracings. The premature auricular beat (P' and a') is followed by a ventricular contraction ($R' T'$, c') and the small premature radial beat (*).

quent, as in the slowing after exertion or after retiring to bed.

Diagnosis.—A premature contraction is recognized at the pulse by an interruption in its regularity, either as a small beat occurring quickly after a normal radial beat and followed by a pause, or as an intermission if the small beat is imperceptible (so-called "dropped beat"). In either case the premature beat will be recognized by auscultation as a feeble *lub-dupp* or *lub* early in the pause at the wrist. The intermission may be very occasional, or it may recur periodically after each normal beat, or after every second, third, or fourth regular beat. Similar intermissions are far less commonly due to heart-block, and silence on auscultation during the pauses at the wrist will decide when this is the cause. If prema-

ture contractions are numerous, the resulting irregularity may simulate that of auricular fibrillation, but a few arm exercises will increase the heart-rate and dissipate premature contractions, while rendering the grosser irregularity of fibrillation more obvious.

On the radial curve the small premature beat is usually visible, and the subsequent pause is not compensatory, i.e. the period between the preceding and succeeding beats is less than two normal pulse periods. On the jugular curve is seen a premature auricular wave (a'), followed by a carotid wave (c') corresponding to the small radial beat. On the electrocardiogram the premature and inverted deflection P' is similarly followed by a ventricular complex of the usual form ($R' T'$). (Fig. 35.) (2) Premature *ventricular* contractions arise in the ventricle, and are more common than the auricular variety. On the radial curve the small beat is seen, or it may be wanting if the abnormal contraction is too feeble. In any case, the period between the preceding and succeeding beats is here equal to that of two

rhythmic beats because the auricular rhythm is undisturbed. On the jugular curve the premature carotid wave (c') is seen corresponding with the small radial beat, if this shows at all. The electrocardiogram records a remarkable diphasic deflection which is characteristic of an abnormal impulse arising in the ventricular muscle (Fig. 36).

The other varieties of premature beats (such as interpolated, nodal, blocked) are rare, and graphic methods are required for their identification.

Prognosis.—As Mackenzie states, when premature contractions are the only abnormal sign, the prognosis is good, and when they are associated with other signs the prognosis should be based on these other signs. When they appear for the first time during an acute in-

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fection, such as rheumatic fever, they are possibly associated with an acute myocarditis. Auricular premature contractions in large numbers have been known to precede the onset of auricular fibrillation or flutter.

Treatment.—If premature contractions are casually found in a patient, he should not be told of them. When they give rise to symptoms, some provocative cause may be found such as over-smoking, indigestion, or very commonly a disordered nervous system. The treatment indicated may be that of an underlying neurasthenia. Bromide may be of value in these conditions, but digitalis is useless and may even induce them, as in the well-known

Symptoms.—The symptoms of heart failure of some severity are generally present. Breathlessness is often severe, anginal pain is common, and enlargement of the heart is seldom absent. It is a common finding in patients over 50 with the following conditions: (1) Arterio-sclerosis with high blood-pressure. (2) Chronic renal disease, often with uræmia. (3) Serious myocardial disease. In any of these it is a special sign of exhaustion of contractility with heart failure. (4) Paroxysmal tachycardia, including auricular flutter. Here it may be no more than a sign of temporary myocardial exhaustion directly due to the excessive ventricular rate.

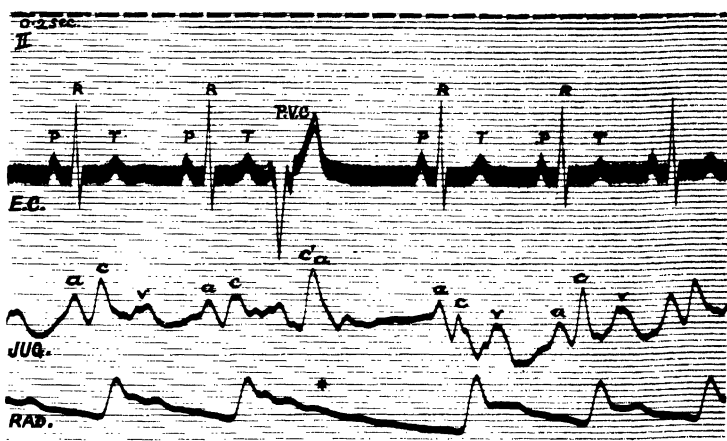


Fig. 36.—Premature ventricular contraction. Simultaneous electrocardiographic, jugular, and radial tracings. The rhythm is normal until the premature ventricular contraction (P.V.C.), which in this instance produces no radial beat, but only an intermission at the wrist.

“coupling.” As the rule is for them to persist in some degree indefinitely, patients should be assured that they are not a sign of heart disease.

PULSUS ALTERNANS

Pulsus alternans is the alternation of large and small beats in a regular pulse and with a normal cardiac rhythm.

It is due to exhaustion of contractility of the heart-muscle, showing itself by a systolic output first large, then small. No pathological lesion is characteristic, but advanced organic disease of the cardio-vascular system is generally found in cases which show this sign.

Diagnosis.—Palpation of the radial pulse reveals alternation only when this is extreme. Further, true pulsus alternans is difficult to distinguish by the finger from a *pulsus bigeminus* or coupling due to premature contractions. In fact, a radial tracing is the only satisfactory means for its identification. The diagnostic feature is the alternation of large and small beats which are *equidistant* (Fig. 37). Pulsus alternans is accentuated after the disturbance due to a premature contraction, or it may not be evident except in the few beats which succeed such an abnormal contraction. Again, a tracing which presents no alternation

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when the rate is moderate may show it clearly when the rate is high in the same individual, e.g. in a paroxysm of tachycardia.

Pseudo-alternans may arise from: (1) Premature contractions when these recur regularly after every normal beat (Fig. 38). (2) Auricular flutter where alternating grades of

The importance of distinguishing true pulsus alternans from pseudo-alternans, which has no prognostic interest, will be evident. Where alternation arises merely from transient exhaustion of contractility due to extreme tachycardia, as in a paroxysm, little importance is attached to it.

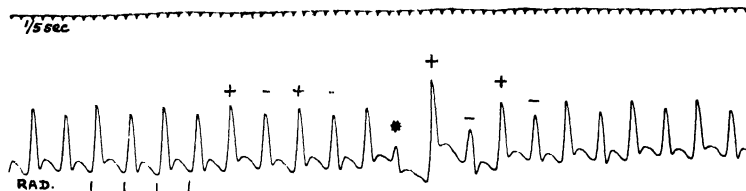


Fig. 37.—Pulsus alternans. Radial tracing which shows equidistant beats, alternately large and small. The alternation is accentuated after the single premature contraction (*).

block (e.g. 3:1 with 2:1) may produce a radial pseudo-alternans hard to distinguish without an electrocardiogram. True pulsus alternans may, however, occur with auricular flutter (Fig. 8, Vol. I, p. 143). (3) Rapid respirations when these happen to affect the size of alternate radial beats (respiratory pseudo-alternans). (4) Extreme diastolic of

Treatment.—This should be directed to the underlying condition, myocardial, hypertensive, or renal. Rest is imperative. Digitalis is capable of inducing alternation as well as other disorders of the heart-beat; but it is not contraindicated because alternation is present, and sometimes it proves useful in alleviating the symptoms of failure. A general

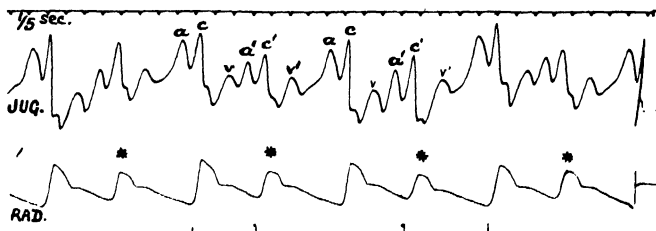


Fig. 38.—Pulsus pseudo-alternans. Coupling of radial beats due to a premature auricular contraction (*) after each normal beat. The radial beats are of unequal height, but they are not equidistant. (cf. Fig. 37.)

the pulse, easily recognized because the diastolic wave is unattended by any heart-sound.

Prognosis.—Persistent pulsus alternans with a moderate heart-rate is, even considered alone, a sign of great prognostic value. It is of such grave import that few patients live for two years from the date of its recognition. Cheyne-Stokes respiration is not uncommon in association. The slighter form of alternation seen only after a premature beat has undoubted significance, though it is not so serious as the persistent form.

anaesthetic should never be permitted when alternation is present unless it is absolutely essential.

Auricular fibrillation and auricular flutter are dealt with under their respective titles.

JOHN PARKINSON.

HEART-BLOCK.—A partial or complete blocking of the normal stimulus from auricle to ventricle, the result of interference with conduction through the auriculo-ventricular bundle.

HEART-BLOCK

The Stokes-Adams syndrome is the combination of recurrent syncopal or convulsive attacks with a slow pulse due to partial or complete heart-block.

Etiology.—Heart-block may occur at any age, but it is more common in adult and later life than in childhood; it is more frequent in males than in females.

Acute heart-block may occur in acute rheumatism, and in 50 consecutive cases of this infection it developed to some degree in 15 (30 per cent.). It has been recorded during diphtheria, influenza, and other acute infections, and in general septic infections, notably septic endocarditis.

Chronic heart-block is commonly a late result of syphilitic infection of the heart, especially when the block is complete. Another frequent cause is antecedent acute rheumatism or chorea, diseases which are responsible particularly for the slight or moderate grades. Of less definite etiology are cases arising from local pathological lesions, the result of arterial disease or other degenerative changes due to age.

Pathology.—By experiments on dogs it has been shown that graduated crushing of the auriculo-ventricular (*A-V*) bundle produces heart-block of a degree commensurate with the injury; complete section of the main bundle invariably results in complete dissociation of the auricle and ventricle.

Pathological findings in man confirm this experimental work. Although disease may slowly invade part of the bundle without block, no case has been reported where conduction was unaffected after a lesion had entirely destroyed its continuity. Conversely, the heart of a patient with heart-block during life shows on histological examination at death an adequate lesion of the *A-V* conducting tissue. As the basis of *acute* block, acute myocarditis is found involving the bundle which lies embedded in the myocardium. As the basis of *chronic* block, gumma or diffuse syphilitic infiltration and fibrosis are common findings. Apart from syphilis, localized fibrotic, degenerative, or calcareous changes affecting the myocardium may implicate the bundle, changes often secondary to narrowing or occlusion of small branches of the coronary arteries.

Stimulation of the vagus, e.g. by pressure in the neck, can determine a transient block, for the vagus conveys inhibitory fibres to the *A-V* conducting system, and it is by stimulation of these fibres that digitalis may temporarily induce or accentuate block.

Symptomatology.—Heart-block is often discovered clinically in the absence of any symptoms attributable to it. In fact, it should be considered in its significance as a sign unaccompanied by special symptoms, excepting only where it is the foundation of the grave syncopal attacks first described by Adams and Stokes in association with a slow pulse. It is only in a minority of patients showing heart-block that such symptoms ever arise.

Stokes-Adams syndrome.—When block is extreme or complete, and few if any of the auricular impulses reach the ventricle, life would cease were it not possible for the ventricle itself to initiate contractions. This independent action of the ventricle is a slow one, commonly 30-40 a minute, and, until it is properly installed, the rate may fall even lower and render the patient liable to attacks of cerebral anaemia. This is manifested by dizziness, syncope, convulsions, coma, even death, depending on the duration of standstill of the ventricle. In exceptional cases the attacks result from short runs of ventricular tachycardia with an imperceptible pulse equally incapable of maintaining the cerebral circulation. The patient is usually a man well advanced in years who, for some time, has displayed symptoms and signs of arterial and myocardial disease. He may have felt nothing more than increased breathlessness on exertion, but often he will have suffered much from dyspnoea, left thoracic pain, palpitation, insomnia, and dizziness. Then, without any relation to the severity of these chronic symptoms of cardiac failure, syncope occurs—perhaps frequently recurring to an alarming extent. The attacks vary in degree from momentary loss of consciousness to convulsions and coma, or these may alternate. The face is pallid or cyanotic, the breathing stertorous or of Cheyne-Stokes type, and the extremities are cold. The pulse is remarkably and persistently slow, 30-40 a minute, perhaps falling under observation to 10-20, and ceasing entirely with the loss of consciousness. The pulse at the wrist corresponds with the ventricular beats, and therefore with the heart-sounds at the apex, but in the neck are seen more frequent pulsations imparted by the auricle, which is beating independently. The arteries are thickened and tortuous; the systolic blood-pressure is often high—150 to 250 mm. Hg—apart from the actual attacks. Cardiac enlargement is usually present, but no

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distinctive sounds or murmurs are heard, except the muffled beats of the auricle between and periodically coinciding with the ventricular beats. The urine is often scanty and albuminous.

Myocardial disease and heart-block.—The striking symptoms of Stokes-Adams disease have been described above as dependent upon severe or complete heart-block, though many with equal block escape such attacks. Slight and moderate degrees of block are far more common, and may produce no character-

The grades of heart-block.—(1) Delayed conduction or slight heart-block, only to be recognized by graphic records. (2) Partial heart-block, in which certain auricular impulses are fully blocked and fail to effect a ventricular contraction. The result of such partial block is a "dropped beat" or pause at the wrist (Fig. 39). If these pauses recur periodically, e.g. 1 in 3, or 1 in 2, the radial pulse will be correspondingly slow (40-50) and, with some ratios, irregular. (3) Complete heart-block, where no impulses pass from auricle to ventricle. The

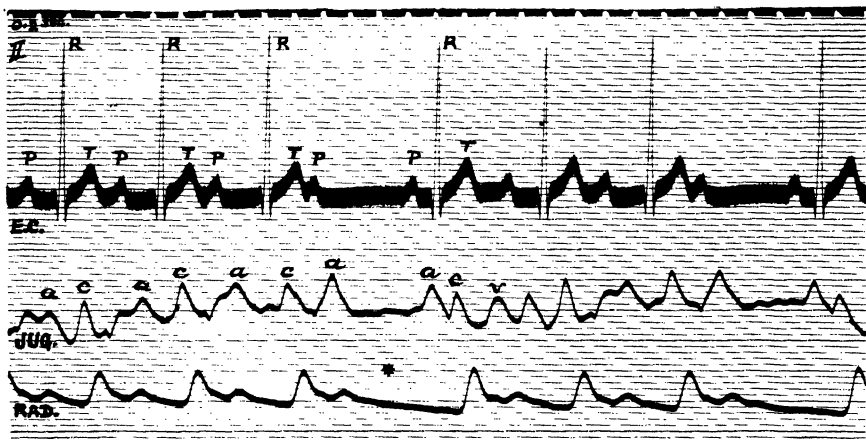


Fig. 39.—Partial heart-block. Triple record. The interval between auricular contraction (P on electrocardiogram, and a on jugular tracing) and ventricular contraction (R on electrocardiogram, and c on jugular tracing) is greater than the normal—0.2 sec.—and this P-R or a-c interval gradually increases until an auricular beat fails to produce a ventricular beat; hence the pause at the wrist (*).

The sequence of increasing block is then repeated.

istic symptoms. But they have considerable significance as a physical sign; indeed, block is the only direct sign of myocardial disease. There may be symptoms, and serious ones, but they denote impaired efficiency of the heart-muscle and are an expression of the myocardial disease. Conspicuous among such symptoms are breathlessness on exertion, left thoracic pain, palpitation, and general weakness. In a proportion of cases mitral stenosis or other rheumatic valvular disease is associated. The grade of heart-block is no guide to the extent or severity of the myocardial lesion; while, on the other hand, myocardial disease commonly runs its course without any block.

auricle beats independently at its normal rate, about 70, the ventricle at its slow rate of 30-40 a minute (Fig. 40). Dissociation has occasionally been reported at higher ventricular rates.

Vagal heart-block.—Apart from acute infections, high grades of block have occasionally proved to be transient. In such cases a vagal origin has been assumed, especially where atropine temporarily abolished it.

Diagnosis.—If, in an elderly man with arterio-sclerosis, repeated attacks of unconsciousness occur in association with a pulse-rate persistently below 40 a minute, the diagnosis of Stokes-Adams syndrome may be made. In any patient, whether syncope occurs or not, a regular pulse persistently maintained at 30-40

HEART-BLOCK

a minute is presumably due to complete heart-block.

Simple bradycardia with a rate of 50-60 occurs in certain persons, especially in the aged, but exertion increases the rate in such cases and a graphic record will prove that block is absent.

Partial heart-block may produce an occasional intermission at the wrist, or, if this recurs frequently, an irregular pulse (perhaps grouped in twos or threes) at a rate of 40-50 a minute. In either case the diagnostic feature of heart-block is *silence* on listening at the apex during the intermission. By auscultation,

waves are not succeeded by the appropriate *c* wave, a ventricular beat must have been blocked and a corresponding pause will be evident in the radial tracing (Fig. 39). In this way the radial pulse in partial block may even be halved (2:1 block) by the interception of every other auricular impulse. In the last degree, equidistant *a* waves are seen in the jugular curve with none but a fortuitous relation to the *c* waves; the two chambers are dissociated, the pulse is slow (30-40) and regular, and complete heart-block is present (Fig. 40).

In an electrocardiogram the electrical varia-

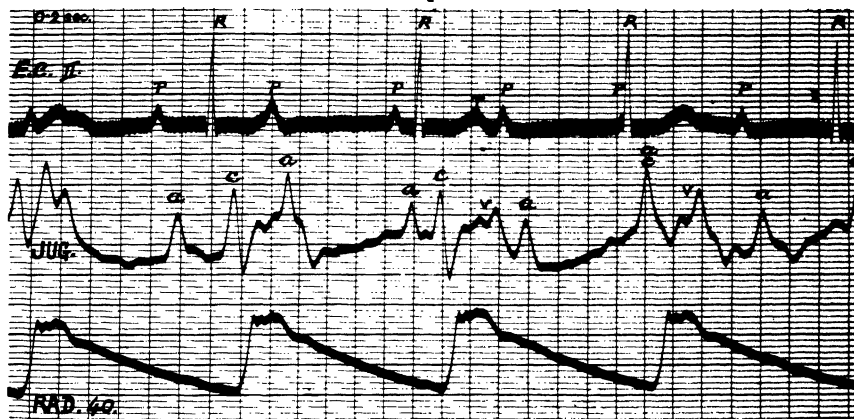


Fig. 40.—Complete heart-block. Triple record. The auricular contractions, represented by P on the electrocardiogram and *a* on the jugular tracing, are completely dissociated from the ventricular contractions, which are indicated by R (or R-T) on the electrocardiogram, *c* on the jugular tracing, and by the radial beats at a rate of 40 a minute.

therefore, an intermission or “dropped beat” due to heart-block is readily distinguished from the commoner intermission due to a premature contraction, for the latter gives a feeble premature *lub-dupp* at the apex during the pause at the wrist.

Graphic methods.—By polygraph or electrocardiograph the presence and degree of heart-block can be demonstrated. In the jugulo-carotid curve of the polygram there are only two waves concerned, *a* the auricular wave, and *c* the carotid wave indicating ventricular contraction. The normal sequence is *a-c* within $\frac{1}{2}$ sec., so that if the *a-c* interval is prolonged beyond this we infer delay in conduction, i.e. slight heart-block. If, in addition, certain *a*

tion accompanying auricular contraction is called P, and that coinciding with the onset of ventricular contraction is called R. Otherwise the indications of heart-block are the same as described for *a* and *c* waves respectively (Figs. 39 and 40).

Prognosis.—In a patient first seen during the attacks peculiar to the Stokes-Adams syndrome, the pallor and unconsciousness will sufficiently ensure a serious outlook on his complaint; and he may die in any attack, though it is seldom he succumbs in the first. Many cases recover and sooner or later attain absolute freedom from attacks. Even if convulsions and coma occur and attacks recur frequently for days, the patient may survive and live for months

HEART-BLOCK

or years free from further recurrence and in fair health. Of course, with the rarest exceptions, the heart-block persists, and with it the cardio-vascular disease of which it is a local sign. Hence the prognosis will depend upon the power of the myocardium to maintain an efficient circulation; but of this efficiency the degree of block is not an index. It is best ascertained from the patient's capacity for exertion without distress. This should not merely be compared with that expected in a man of his years, but also with his own capabilities so many months and so many years before. Such consideration of symptoms will help most in prognosis. Of the physical signs, those indicative of great enlargement of the heart, extreme arterio-sclerosis, and secondary renal inadequacy are the most serious. Œdema of the legs may be the first direct sign of impending cardiac failure. In most cases of complete heart-block, though not nearly in all, the associated myocardial disease will limit the expectation of life to a few years.

The acute heart-block arising during the course of acute rheumatism and other infections is almost invariably transient, though the injury sustained may show itself later in minor permanent forms of chronic block.

Treatment. (1) *Stokes-Adams syndrome.*—In the presence of attacks the patient should lie with head low and legs raised or tightly bandaged. The body must be kept warm by abundant clothing and hot bottles. Oxygen should be administered without stint in cases showing cyanosis. The subcutaneous injection of $\frac{1}{10}$ — $\frac{1}{20}$ gr. of atropine sulphate is advisable to nullify the vagal impulses which may possibly impede the ventricular slowing in an occasional case; it may be repeated as necessary if the symptoms abate or the pulse-rate increases as a result. Digitalis may be administered if there are accompanying signs of failure, such as œdema; it does not further reduce the slow rate.

In a case with a syphilitic history or giving a positive Wassermann test, a course of mercury, ung. hydrargyri 1 dr. daily, should be practised for six weeks. Iodide $\frac{1}{2}$ –1 dr., well diluted, may be given daily for several months, avoiding only iodism. Salvarsan preparations will often be contraindicated in old arterio-sclerotic patients, but if enterprised, a small dose intravenously should first be used, such as 0.3 grm., to be followed by four doses of 0.6 grm. at intervals of a week.

(2) *Acute heart-block.*—The development of

HEMIANOPIA

block during acute rheumatism, diphtheria, or other infections should be accepted as a sign of acute myocarditis necessitating prolonged rest in bed and a very gradual return to physical exertion.

(3) *Chronic heart-block.*—As this gives rise to no characteristic symptoms, except in cases with Stokes-Adams syndrome, treatment is directed to the underlying arterial and myocardial disease. Syphilis is treated as suggested above. In general arterio-sclerosis, iodide may be given in doses of 15–30 gr. daily, with intermissions. When symptoms and signs of heart failure appear, especially with œdema, digitalis should not be withheld, though it may intensify a partial block. JOHN PARKINSON.

HEAT-EXHAUSTION (*see* SUNSTROKE).

HEAT-STROKE (*see* SUNSTROKE).

HEBEPHRENIA (*see* DEMENTIA PRÆCOX).

HEBRA'S PRURIGO (*see* PRURIGO).

HELMINTHIASIS (*see* INTESTINAL WORMS).

HEMERALOPIA (*see* DAY BLINDNESS AND NIGHT BLINDNESS).

HEMIANÆSTHESIA (*see* NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF).

HEMIANOPIA.—Blindness of the corresponding halves of the visual fields in both eyes, due to lesions affecting the conducting paths or visual centres at or behind the chiasma where the two optic nerves undergo a partial decussation. A lesion at the chiasma itself often produces loss of vision in both temporal fields (bitemporal hemianopia), as it is liable to injure the decussating fibres that convey impulses from the nasal half of each retina. Lesions of the optic tracts or centres behind the chiasma produce homonymous hemianopia on the opposite side—that is, blindness in the temporal half of the field of the contralateral eye, and in the nasal half of the homolateral eye.

The blindness in hemianopia due to a lesion in front of the occipital cortex, in the optic tract, primary optic centres, or optic radiations, is generally limited by vertical lines through the fixation points, but only portions of the homonymous fields may be lost, particularly in small lesions of the parietal lobe that involve the optic radiations. When the visual area in the occipital cortex is disturbed, the hemianopia varies with the extent and situa-

HEMIHYPERTROPHY, CONGENITAL

tion of the lesion. The visuo-sensory cortex is distributed round the calcarine fissures on the inner aspects of the occipital lobes, from the junction of the calcarine with the parieto-occipital fissures to the posterior poles of the hemispheres. Macular or central vision is represented at the posterior pole, and the successive concentric zones of the visual fields from the fixation point outwards are represented in the same order from behind forwards along the calcarine fissure. Further, the cortex of the upper portions of the calcarine areas corresponds to the upper halves of the retina, and the lower to the lower. Consequently, if we were to unfold the calcarine fissure by pulling its lips apart, the line forming the deepest part of the fold would correspond in function to the horizontal meridians of the opposite halves of the visual fields, the cortex above this line to the lower halves of the fields, and that below this line to the upper portions of the fields. Owing to this areal projection of the retina on the visual cortex, small lesions in the calcarine region may produce loss of vision in limited areas of the opposite homonymous fields. The most common of these defects are the quadrantic hemianopias, in which the blindness is roughly limited by the horizontal meridians.

The fact that many lesions affect both radiations and cortex adds considerably to the difficulties found in the interpretation of visual defects due to cerebral lesions.

The Wernicke pupillary reaction occasionally proves useful in differentiating between a hemianopia produced by a lesion of the tract or primary optic centres and one situated in the optic radiations or occipital lobe. When a disturbance producing hemianopia is in the former region, the pupil fails to react to light thrown on the blind portion of the retina. The test has to be made with care, and on the whole is of no great value. LESLIE PATON.

HEMIATROPHY (*see* HEMIHYPERTROPHY, CONGENITAL).

HEMIATROPHY OF FACE (*see* FACIAL HEMIAMATROPHY).

HEMIORANIA (*see* MIGRAINE).

HEMIHYPERTROPHY, CONGENITAL.

—Hypertrophy limited to one side of the body has occasionally been noticed within a few months or years after birth. The affected side continues to grow more quickly than the other,

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so that a marked asymmetry results. The limbs on the hypertrophied side are both longer and more developed than the normal, and in a typical case all the structures on that side, including one half of the tongue, and the corresponding tonsil, salivary glands, kidney, suprarenal gland, and testicle, take part in the overgrowth. When the hypertrophy is left-sided the patient is usually left-handed. The causation is not known, but a central nervous origin is suspected. In other cases the hypertrophy is crossed in distribution, the leg being affected on one side of the body and the arm on the other. Possibly one limb only may be affected, but such cases are capable of other interpretations.

This unusual condition must be distinguished from the hemiatrophy (more correctly styled hypotrophy) which is either congenital in origin and due to cerebral lesions before birth, or results from similar lesions during childhood. It is also simulated by hemiatrophy occurring after full bodily development, and generally associated with scleroderma. Cases in which more fat occurs on one side of the body than on the other may be imperfect examples of either hemihypertrophy or hemiatrophy.

Finally, hemihypertrophy must be separated from trophædema, in which a solid œdema occurs in one or more limbs; and from enlargement of the limbs due to dilated congeries of veins or lymphatics (pseudo-elephantiasis).

No satisfactory treatment is known.

FREDERICK LANGMEAD.

HEMIOPIA (*see* HEMIANOPIA).

HEMIPLEGIA.—A condition of weakness or paralysis of the movements of one side of the body, due to destruction of the cortical motor centres of one hemisphere, or to interruption of one pyramidal tract above the cervical enlargement. It may be produced by lesions of vascular origin, as hæmorrhage from or occlusion of an artery, by a tumour or abscess which compresses or invades the motor cortex or its efferent fibres, by their involvement in encephalitis or their injury by trauma. Arterial spasm may be responsible for transient attacks. Hemiplegia is consequently a symptom rather than a disease; the disease is the pathological condition that has caused it. As the pyramidal tracts decussate in the posterior portion of the medulla oblongata, the loss of power is on the opposite side to that of the

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lesion in the brain when the disease lies above this level.

Symptomatology.—In the early stage the loss of power is essentially a flaccid palsy—i.e. the muscles are flabby and toneless, the affected limbs when moved passively offer little or no resistance, and if raised fall immediately and inertly to the bed. The palsied cheek flaps in and out with each respiration. When the onset is sudden there is occasionally a phase of *early rigidity* which lasts a few days only. *Late or permanent rigidity* usually begins to develop three or four weeks after the stroke, and the palsy then becomes spastic. If the onset is gradual, as when due to a cerebral tumour, rigidity appears much later.

The degree of paralysis varies in different groups of muscles and with the stage of the illness. In the severer cases the loss of power is at first usually complete in the arm, but there is always some return of movement at the shoulder, and feeble flexion of the elbow generally becomes possible. The paralysis of the wrist and fingers, on the contrary, often remains complete. In milder cases the rule is that the proximal muscles are less affected than the distal; there may be little feebleness of the movements of the shoulder and elbow though the fingers are paralysed or very weak. Certain movements are also liable to suffer more than others; abduction of the shoulder is always much weaker than adduction, extension of the elbow than flexion, and the extensors of the fingers are invariably more seriously involved than the flexors.

In the lower limbs also the movements of the distal segments are affected more than those of the proximal. It is rare to find no power of flexion and extension of the hip after the first few weeks, though the ankle movements are lost, or very feeble and restricted in range. Loss of the individual movements of the toes, which can be then moved only *en bloc*, is, as a rule, the most obvious sign of a mild or recovering paresis of this limb. The affection of the trunk muscles is always less obvious than that of the limbs; a paresis of the abdominal muscles may make it difficult for the patient to sit up, and paresis of the erector spinæ allows him to fall or slide over to one side. As Hughlings Jackson pointed out, the excursions of the affected side of the chest are often greater than those of the opposite side in ordinary respiration, since the bulbar centre which regulates the automatic movements of respiration is freed

from cerebral control, while in voluntary expansion of the chest this side moves less than the normal. The relative escape of the trunk movements is usually explained by Broadbent's hypothesis, that muscles which habitually act symmetrically with the corresponding muscles of the other side receive cortical impulses from both halves of the brain, and consequently when a lesion interrupts the one path the other hemisphere can still supply stimuli for voluntary movement.

It is for the same reason that the muscles of the upper part of the face, the frontalis and corrugator, are never completely paralysed. The orbicularis palpebrarum usually escapes to such an extent that the patient can close his eyes comfortably, though there is generally an unmistakable weakness when he is asked to screw them up tightly. On the other hand, the movements of the cheek and lips are considerably affected; in showing the teeth this corner of the mouth is raised and retracted less than on the normal side, the naso-labial fold is less pronounced, and the muscles concerned in the act tire quickly if the effort be maintained. In attempting to whistle or blow out the cheeks the air escapes between the lips; also, food collects between this cheek and the teeth. When the palsy is severe, saliva and fluids taken into the mouth may dribble from the affected corner. The tongue when protruded deviates towards the paralysed side. On the other hand, the muscles of mastication, the masseter, temporal, and pterygoid, are not appreciably affected, and, unless the causal lesion involves the brain-stem, none of the ocular muscles are paralysed. When, however, there is hemianopia the eyes are usually deviated towards the unaffected side, and lateral movement in the opposite direction is incomplete or requires undue effort. There may be a transient difficulty in swallowing in the early stages of a severe attack, and the articulation of words may be affected too. These symptoms are more common in left- than in right-sided hemiplegia.

In recovery, of which some evidence is generally found in the second or third week at least, the return of power occurs in the reverse order to the degree of paralysis. The movements of the face, and then those of the leg, begin to improve, while the arm, or its distal segments, may remain paralysed. Many patients walk again even when the cerebral destruction is extensive, while even slight lesions may leave the hand useless. It is

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always the finer and later-acquired functions that suffer most; the skilled mechanic or the musician can rarely resume his former occupation with his original skill, no matter how complete the recovery may be.

A further complication is the onset of *late rigidity*. This is due to the development of an excess of tone in the paretic muscles owing to the removal of the inhibitory control by which the forebrain checks the spinal and bulbar reflex mechanisms. Rigidity is an exaggeration of the normal tone of the muscles. On its appearance the limbs become stiff and resistant to passive movement, and it restricts the range and makes it more difficult for the patient to execute the movements that he can innervate. As voluntary effort may excite the reflex centres, movements that the patient does not desire may occur. The rigidity predominates in certain groups of muscles, as in the adductors and flexors of the arm and the extensors of the leg; it consequently tends to keep the limbs in certain attitudes, and *contractures*, or organic shortening of the muscles, are then liable to occur. This leads to fixation of the limbs in positions from which they cannot be moved by any moderate force. The arm is usually adducted at the shoulder and flexed at the elbow, fingers, and wrist, while in the leg the shortening of the calf muscles prevents dorsiflexion at the ankle, and the heel does not come to the ground in walking.

The modification of the *reflexes* is constant and characteristic. The arm-jerks, knee-jerk, and ankle-jerk are exaggerated on the palsied side, and clonus can usually be elicited, especially at the ankle and knee. Contractures of long standing may make it difficult to elicit these reflexes. The abdominal reflexes are, on the other hand, absent, and stimulation of the sole evokes an extensor response—i.e. Babinski's sign—on the affected side.

The state of *sensation* varies. In many cases of severe motor paralysis no disturbance can be detected; in a small proportion there is considerable loss, while in the majority it is only by careful examination that any alteration can be discovered. This variability depends on the site of the lesion. As the perception of touch, pain, and temperature depends primarily on subcortical centres, and especially on the optic thalamus, these forms are never affected severely by cortical lesions. The presence of complete anæsthesia in such cases always suggests hysterical complications. But the discriminative properties of sensation, as

the localization of stimuli, the sense of position, and the appreciation of size, shape, and weight, are cortical functions that suffer when the sensory cortex in the parietal lobe or its afferent fibres are damaged. These disturbances, which are frequently neglected in ordinary examination, are much more important than loss of cutaneous sensibility, since they make the limb useless for all expert or delicate work. Lesions of the posterior limb of the internal capsule, which frequently extend into the optic thalamus, may produce grosser loss of all forms of sensation; and if the fibres of the sensory path are involved in the brain-stem where they run in a compact bundle there may be a still severer affection.

Capsular lesions often produce a *hemianopia* (q.v.), and cortical and subcortical foci in the left hemisphere are often associated with *aphasia* (q.v.). Rarer complications are the presence of severe *pains* in the paralysed side of the body, occurring when the lateral nucleus of the thalamus is involved (thalamic syndrome), and the occurrence of *involuntary movements*, as tremor, post-hemiplegic chorea, and athetosis; the latter are usually associated with lesions of the basal ganglia and midbrain. Post-hemiplegic *epilepsy* is not uncommon; it occurs either as localized spasms of the affected limbs, or as generalized convulsions with loss of consciousness.

Trophic disturbances are not so prominent as in paraplegia. The bedsores which may develop in an unconscious or severely paralysed patient are due to bad nursing and are avoidable. The paralysed muscles waste only to a slight degree; the skin becomes dry and badly nourished, and the nails grow irregular and lose their gloss. The most important of these changes are the arthritic lesions, which lead to articular roughening and adhesions and to periarticular thickening. All the joints of the affected limbs may be involved, but most commonly the shoulder, the wrist, and the metacarpo-phalangeal. These lesions restrict the range of movement and cause much pain. Arthritis is the most frequent cause of pain in hemiplegia.

The clinical picture of hemiplegia varies with the site of the disease. Cortical lesions tend to cause limited palsies with affection of the discriminative aspects of sensation only, while capsular destructions produce widespread paralysis and grosser sensory loss, often associated with hemianopia. In the upper part of the midbrain the nucleus or root of one of the

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oculo-motor nerves may be involved in addition to the homolateral pyramidal tract, and consequently paralysis of certain of the ocular muscles on the side of the lesion and a hemiplegia of the opposite side result—*superior alternate paralysis*. In the pons the trigeminal roots may suffer, so that we find paralysis of the masticatory muscles and loss of sensation on the same side of the face, with paralysis of the opposite limbs; or the facial nerve or its nucleus may be damaged so that a flaccid and atrophic palsy of the face is associated with a spastic weakness of the opposite side of the body (the *Millard-Gubler type*). Lesions lower in the bulb may cause paralysis of the tongue, palate, or larynx, with an alternate hemiplegia. In disease of the brain-stem the homolateral unparalysed limbs may be ataxic owing to involvement of part of the cerebellar system. Unilateral injury or disease in upper segments of the cervical cord often produces a spastic paralysis of the limbs and trunk of the same side, since the pyramidal tract has already decussated at this level, but the functions of the cranial nerves are not affected and there is usually loss of pain and thermal sensibility on the opposite side (the *Brown-Séquard syndrome*).

Differential diagnosis.—Hemiplegia is a symptom of disease of the upper motor path. A study of the distribution of the paralysis, rigidity, and contractures, and especially of the reflex changes, can rarely leave any doubt of its nature. In diagnosis the question of *hysteria* is the most important, as hysterical hemiplegia is not uncommon. The distinctive features are the mode of onset, the absence of any adequate cause as vascular or cardiac disease, and the state of the reflexes, which are never abnormal in hysteria. It may be difficult to elicit the plantar reflex, but the essential point is that an extensor response, which is almost invariable in hemiplegia, can never be obtained. Further, positive signs of hysteria, as complete or irregular hemianæsthesia, restriction of the visual fields, and abnormal suggestibility, can confirm the diagnosis. Finally, hysterical hemiplegia is flaccid; and if the limbs oppose resistance to passive movement it is due to an active contraction which can easily be distinguished from true rigidity.

Hemiplegia is occasionally a symptom of *uræmic states*, and is then due to structural or functional affections of the motor system or to transient spasm of the arteries. More

difficulty may be experienced in its differentiation from certain cases of chorea and paralysis agitans. Many cases of *chorea* begin with loss of power on one side, and that disease should always be suspected when this symptom develops gradually in a child or a young pregnant woman. This paralysis is always flaccid, an extensor response can never be obtained, and the characteristic restless movements can generally be observed, though perhaps only in the tongue. In unilateral *paralysis agitans* the uselessness of the limbs develops gradually, the peculiar stiffness of the muscles is generally more prominent than their loss of power, the reflexes are unaltered, and the observation or the history of tremor settles the diagnosis.

Treatment.—The proper treatment of hemiplegia, no matter what its origin, is extremely important in the early stages, though often neglected. Its aims must be (1) to keep the muscles in as natural a state of nutrition as possible, (2) to prevent the formation of contractures and joint-changes which may limit the range of the movements that the patient can perform, (3) to encourage and make the most of any voluntary movement that returns, (4) to re-educate as far as possible the functions of the affected limbs.

Massage and passive limb movements are essential when the limbs are badly paralysed. This treatment should be begun in the first week and applied once a day at least; the massage may be vigorous, with deep kneading of the muscles; the passive movements should be through the full possible range at every joint. Electricity is not necessary and is often harmful, as it tends to excite reflex contractions and increase the rigidity. To avoid the formation of contractures and joint-changes it is, in the first place, necessary to prevent the limbs from lying for long in any one position, as the muscles tend to shorten and the joints to become fixed in the position they are allowed to assume. The elbow, for instance, will become fixed in flexion if the arm constantly lies flexed, and in extension if the elbow is permanently extended. There is consequently a better chance of a useful limb if it is frequently moved from one position to another. As rigidity is essentially a reflex phenomenon excited by peripheral stimuli, the limb should be preserved from cold, draughts, and other excitations by being wrapped in cotton-wool or enclosed in long stockings; this is particularly important in the arm. Frequent passive movements and massage around the

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joints are the only means of avoiding the occurrence of disabling arthritic changes.

Many hemiplegics readily pass into a passive, resigned state, and rely on massage and electricity to help their recovery. It is therefore extremely important to insist to the patient that any movements he can make unaided are much more valuable as a stimulus to recovery than anything the masseur or electrician can do for him. He must be urged to make as perfect as possible any movements he can do and to acquire new ones. Re-education, when persisted in, is frequently most effective. The patient's awkwardness is partly due to the fact that he is satisfied with the movements that cost him least effort. These should be carefully analysed and his errors corrected. He soon begins to walk, for instance, more easily and more naturally when trained to flex his knee and raise his foot properly from the floor with each step. The isolated and delicate movements of the fingers can often be improved to a remarkable degree by appropriate education if any power of movement returns. For this purpose a trained attendant is desirable, but a careful doctor can generally devise and recommend exercises which an intelligent patient can then practise alone.

F. C. PURSER.

HENBANE POISONING (*see* POISONS AND POISONING).

HENOC'S PURPURA (*see* PURPURA).

HEPATIC ABSCESS (*see* LIVER, ABSCESS OF; TROPICAL ABSCESS).

HEPATOPTOSIS (*see* VISCEROPTOSIS).

HEREDITARY ATAXIA (*see* ATAXY, HEREDITARY CEREBELLAR; FRIEDREICH'S DISEASE).

HERMAPHRODITISM.—True hermaphroditism is a condition of congenital deformity in which the sexual glands, or gonads, partake of the characters of both sexes, and are called "ovotestes." The external genitals are imperfectly developed and exhibit features common to both sexes, while the secondary sexual characters may be anomalous, mixed, or partially absent. The true hermaphrodite is, however, extremely rare, and so far only five reported cases have stood the test of microscopical examination.

Almost all cases of so-called hermaphroditism really fall under the head of pseudo-herma-

phroditism, in which the gonads are definitely male or female in structure, while the external and internal genitals are of the opposite sex and imperfectly developed. Inasmuch as sex is determined by the sex gland or gonad, these cases must be regarded as males or females with inadequately developed external and internal organs.

This will be more readily understood when it is remembered that the genital system is developed from the same original foetal structures, by a growth of some elements and a partial suppression of others (proper to the opposite sex), so that in both sexes there is normally a complete representation of the original rudimentary structures, the female characters remaining vestigial in the male, and vice versa. Examples of homologous organs are the penis and the clitoris, the scrotum and the labia majora, Cowper's gland and Bartholin's gland.

If, now, during the processes of development of a fetus possessing a testis, the full growth of the external genitals proper to the male sex fails, the penis may approximate to the size of the clitoris, and the lateral halves of the scrotum may not unite and will thus resemble the labia majora. Further, there may be an excess of development of the Müllerian system of Fallopian tubes, uterus, and vagina beyond the vestigial condition found in the normal male, and yet scarcely reaching the complete stage of the normal female. At first sight such a patient would superficially resemble a female, especially if, as is often the case, the testicles were undescended and the penis hypospadiac, but, strictly, he will be a male on account of the testicular characters of the sex gland.

In a similar fashion, but much more rarely, the gonad is an ovary, with median union of the labia majora and hypertrophy of the clitoris. If there are also small labial hernias, the likeness to a male will be still more striking.

The first type, where the sexual gland is a testis and the external organs appear to be those of the female, is defined as a *male pseudo hermaphrodite*, while the second type is described as a *female pseudo-hermaphrodite*.

The secondary sex characters of voice, hair, adipose distribution, mammary development, and muscular power are very variable, and, in cases of this deformity, may not conform to the apparent sex as indicated by the external genitals. Further, they may be of a mixed type, some being those of the male, others peculiar to the female.

The clinical importance of hermaphroditism lies in its diagnosis, for of treatment there is none, except for the minor condition of hypospadias.

Sex is decided by an inspection of the infant's external genitals at birth, and during childhood a pseudo-hermaphrodite is rarely discovered. It is a development of the secondary sex characters at puberty not in keeping with the child's apparent sex which usually gives rise to the suspicion that an error has been made; or later, when the question of marriage is being considered, the patient may seek advice on account of ill-development of the external organs, or because there has been no menstruation. More rarely, a "female" patient may consult a practitioner on account of sterility, when it may be found on examination that "she" is really a male.

When a patient is seen at or just after puberty the essential requirement is the correct diagnosis of the true sex, in order that he or she may take the proper place in social life. As has been already stated, the only true test of sex is the nature of the gonad, but this can rarely be discovered, except in unusual cases where an apparent undescended testicle is removed at operation and found on microscopical examination to be an ovary. A careful examination of the internal and external genital organs will usually point to which sex their general development inclines; and when the secondary sex characters are mixed or partially absent this is the safest guide for determining the real sex of the individual. In those cases, however, in which the genital organs are not sufficiently distinctive, the secondary sex features may serve as a guide, not so much to the true gonad-sex of the patient as to the proper category for social life. In most of such cases the sexual influence of the gonads is not sufficiently strong to overcome the tendencies of education and training, where these have been counter to the true sex, and such individuals should be brought up according to the indication of the secondary characters.

If it is found that a mistake has been made in the previous training, of which both patient and friends are unaware, it is unwise to alter the training or give information of the error. Marriage should always be most strongly discouraged, as the patient is invariably sterile and physically incapable of entering fully into marital life.

A. W. BOURNE.

HERNIA, ABDOMINAL.—A hernia is a protrusion of a viscus from the cavity in which it is normally contained. Abdominal hernias will alone be considered in this article.

Hernias are named according to the position in which they occur, the inguinal, femoral, and umbilical hernias being the most frequent ones. More rarely, diaphragmatic, obturator, and perineal hernias are seen; whilst a ventral hernia is one appearing through the anterior abdominal wall at some point other than those mentioned.

Etiology. 1. **Congenital.**—Russell and Murray have shown that the most important factor is the presence of a congenital pouch of peritoneum. This is especially so in the case of inguinal hernias, only a very small proportion of these being acquired. Many femoral hernias are also congenital. Diaphragmatic hernias are in most cases dependent upon congenital absence of a portion of the diaphragm, but may be due to rupture of the muscle-fibres from trauma.

2. **Increased intra-abdominal pressure.**—This factor may occasionally be the cause of inguinal hernias in elderly people, and also of femoral hernias. Obturator hernias and some hernias of the mid-line are generally due to increased pressure.

3. **Traction.**—This is not a common cause, but pads of extraperitoneal fat in the linea alba, and perhaps even in the inguinal and femoral regions, may drag out with them, as they increase, a pouch of peritoneum which forms a hernial sac.

4. **Traumatism.**—In courts of law it is very commonly maintained that a hernia may be caused by one severe accident. There is no doubt that in the majority, if not in all, of these cases the peritoneal sac was of congenital origin, although it is possible that such an act of trauma might cause a viscus to descend into the sac. In certain cases operation, especially if sepsis of the abdominal wall has occurred, is the cause of one variety of ventral hernia.

Pathology.—The important constituent of a hernia is the sac, a pouch of peritoneum which is present in nearly all cases. In the congenital types it consists of a portion of peritoneum which, present in the embryo, should be obliterated before birth. It is characterized by its flask-like shape. The narrow portion, the neck, where it is attached to the peritoneal cavity, is often thickened in old-standing hernias and may be almost cartila-

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ginous in firmness. Beyond this the sac widens to form the body, and is then rounded off as the fundus. In acquired cases the neck is much wider and frequently, indeed, is the largest portion of the sac. Such a condition is well seen in direct inguinal hernias.

According to the nature of the contents of the sac, different varieties of hernia are recognized :

(a) **Epiplocele.**—The sac contains only omentum. It may be free or adherent. If adherent at the neck sufficiently to occlude the opening, fluid may collect, giving rise to a hydrocele of the hernial sac.

(b) **Enterocoele.**—Here intestine is alone present in the sac. If omentum be also present the condition is known as an *entero-epiplocele*. The gut within the sac may undergo various changes. Its circulation may be obstructed by the sac or by some structure within or without the sac—a condition ultimately leading to gangrene of the gut. The hernia is then said to be *strangulated*. If only a portion of the lumen is strangulated, the condition is known as a *Richter's hernia*. A *Littre's hernia* is one in which a Meckel's diverticulum is strangulated. The contents may be prevented from passing down the lumen, when the hernia is said to be "*obstructed*." Viscera other than the gut may be contained in the sac, such as the bladder, uterus, Fallopian tube, or ovary.

(c) **Extrasaccular hernia.**—In this case a viscus lies partly or wholly outside the hernial sac, although the latter is probably always present. The extrasaccular viscus is one which within the abdomen has normally only a partial peritoneal covering. As the sac increases in size it drags this viscus through the abdominal walls, and thus the portion which is normally retroperitoneal lies outside the hernial sac. The cæcum, bladder, and iliac colon are the viscera most frequently found in this position.

(d) **Irreducible hernia.**—Apart from strangulation or the presence of an extrasaccular viscus, the contents may be irreducible. The commonest causes of this condition are the formation of adhesions between the various contents or between the contents and the sac wall, and the increase in bulk of the contents after they have been prolapsed.

General symptomatology.—There will be no evidence of a hernial sac until some of the contents of the abdomen have protruded into it.

A *reducible* hernia is characterized by a

variable swelling, often disappearing entirely when the patient lies down, but suddenly appearing when he stands or increases his intra-abdominal pressure. When compressed by the examining hand the contents may be reduced into the abdomen and the swelling disappear. In the case of an epiplocele this reduction will be gradual, but in the case of an enterocoele it will be accompanied with a gurgling sound as the gut is pushed back. In addition to the inconvenience of a lump the patient may complain of a certain degree of pain.

Extrasaccular hernias are generally large, and only reducible in part. A solid viscus such as the uterus or ovary may be felt, and if the bladder be present it may be noticed that the swelling enlarges as the bladder fills and is decreased after micturition.

If *strangulation* occur there will be the onset, usually sudden, of severe sickening pain, followed by collapse, vomiting, and complete constipation. The hernia will be extremely tender and irreducible. If it is unrelieved the vomiting becomes fecal, the collapse more pronounced, and the patient moribund. Occasionally the onset is more gradual, the condition often being mistaken for obstruction. In this case the hernia may become irreducible. There will be steadily increasing pain and, later, the onset of vomiting, the patient then passing into the characteristic condition described above. In the early stages the swelling may be mistaken for an inflamed gland, but the sudden onset, the absence of a source of infection, and the presence of vomiting or constipation should prevent such a mistake. *Obstruction* is rare, being most frequently seen in umbilical hernias containing large gut. It is accompanied by colicky pain, occasional vomiting, and visible peristalsis in the hernia. If unrelieved, the increased pressure of the intestinal contents may lead to obstruction of the circulation at the neck of the sac, and hence to strangulation.

INGUINAL HERNIA.—The hernial protrusion takes place into the inguinal canal. Two main varieties are described. In (a) *indirect* hernia the protrusion occurs through the internal abdominal ring, the neck of the sac being external to the deep epigastric artery. In (b) *direct* hernia the sac is situated internally to this vessel. The former variety is probably always congenital in origin, the latter probably acquired.

Pathological anatomy.—Certain varieties of the *indirect* form are recognized. In the con-

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genital the sac is formed by a persistent portion of the processus vaginalis. If the persistent portion extends only a part of the way along the cord the hernia is known as "*funicular*." If it is continuous with the tunica vaginalis the condition is described as a "*vaginal*" hernia. In the "*infantile*" type there is a second pouch of peritoneum; and, according to the condition of the processus vaginalis, six further divisions of this variety have been described by Lockwood. Of late, doubt has been cast upon this method of formation.

With an undescended testicle the conditions are modified. The processus vaginalis is open in its whole course, and there is a potential hernia. Owing to the position of the testicle the hernial sac may burrow among the muscular layers of the abdominal wall, constituting the so-called "*interstitial*" hernia, when the sac is often bilobed. It is always thin-walled and adherent to the structures of the cord.

The *direct* hernia may be situated internally or externally to the obliterated hypogastric artery, and hence will either pass through or lie externally to the conjoined tendon.

Symptoms. (a) *Indirect hernia*.—Frequently a swelling is found shortly after birth. When the child cries there is a definite bulge in the inguinal canal, or perhaps even a mass which passes into the scrotum. The hernia may be reduced and not return for a long period, giving rise, especially if a truss has been worn, to the false belief that the hernial sac is closed. A similar condition may be seen in females, the gut passing along the canal of Nuck and perhaps entering the labium majus. In other cases no abnormality is noticed until early adult life. A swelling may then follow some muscular effort. It is usually well defined even on its first appearance, thus giving evidence of the previous existence of the sac. If of the funicular type it may be limited to the inguinal canal, when it is often termed a *bubonocoele*. If vaginal it may pass at once down to the testicle, the condition then being known as a *scrotal hernia*. The swelling traverses the inguinal canal; it thus lies well above Poupart's ligament and internally to the spine of the pubis. If uncomplicated it has a well-marked impulse on coughing, and is easily reducible—reduction, if the gut be present, being associated with a characteristic gurgle.

(b) *Direct hernia*.—Hernias of this variety occur later in life. They are more gradual in onset, commencing as a slight bulge which

steadily increases until there is a definite hernia. Pain is more likely to be present, but, owing to the width of the neck, strangulation is not common. The swelling occupies only the inner portion of the inguinal canal and appears to project directly outwards from the abdominal wall, its outer limit not reaching nearly as far as the mid-Poupart point. The hernia can be reduced by direct backward pressure. The different appearances of the swelling in the direct and indirect varieties are best discerned by making the patient cough while in an erect posture. Long-standing indirect hernias may, however, in this respect resemble the direct, the internal abdominal ring being dragged inwards until it is opposite the external ring. Owing to the direct outward projection this variety is more likely to be mistaken for a femoral hernia, but it lies internally to the spine of the pubis.

Diagnosis. (a) *Femoral hernia*.—This variety arises below Poupart's ligament and externally to the spine. The neck of the sac may also be felt passing downwards into the saphenous opening. Since it tends to pass upwards over Poupart's ligament it may at times be difficult to distinguish from a small direct inguinal hernia.

(b) *Encysted hydrocele of the cord*.—If the hydrocele is situated high up in the cord it may lie well within the inguinal canal, from which it is expressed when the patient coughs or otherwise raises his intra-abdominal pressure. The swelling therefore appears to have an impulse. It is not, however, completely reducible. It is fixed when the testicle is pulled upon. It is globular in shape, its upper limit can usually be reached, and it is often possible to make out definite fluctuation.

(c) *Vaginal hydrocele*.—Hydroceles, especially in young children, occasionally extend some distance up the cord, and in this case may have an impulse and thus be mistaken for a hernia. The upper limit can, however, be usually reached and definite fluctuation obtained. This sign is of greater value than the presence or absence of translucency, as the latter is present in some hernias and absent in many hydroceles.

(d) *Lipoma of the cord*.—A lipoma of the cord is very prone to be mistaken for an irreducible omental hernia. It occupies the line of the canal, has an impulse, and appears to be partly reducible. Not uncommonly it is associated with a small hernial sac. It is often not diagnosed until operation.

HERNIA, ABDOMINAL

(c) *Enlarged inguinal glands.*—With this condition there is no impulse, the history of the onset is usually shorter, there is greater tenderness, and a primary focus can often be seen. Not uncommonly the condition is bilateral.

FEMORAL HERNIA.—The protrusion occurs through the crural canal, internally to the femoral vein and externally to Gimbernat's ligament. The hernia passes at first directly downwards, then forwards through the saphenous opening and upwards on to the abdominal wall. Rarely, the sac may be in front of, or external to, the femoral vein. It is more common in females than in males, but even in them is not so common as is an inguinal hernia. It is rare in children, but, in spite of this, there is an increasing amount of evidence to show that it is dependent upon the presence of a congenital sac.

Symptoms.—A swelling which may have a definite impulse is found in the femoral region. It appears to come directly forwards, and, although the greater part of it may lie above Poupart's ligament, on attempts at reduction it is found to emerge from below this structure; or the neck of the sac may be felt in the saphenous opening lying below Poupart's ligament and externally to the spine of the pubis. Very frequently the sac contains only omentum. It is commonly irreducible. Owing to the rigidity of the surrounding structures and the narrowness of the opening, strangulation often occurs and may be the first evidence of the presence of a hernia.

Diagnosis.—The points of distinction between femoral and inguinal hernias have already been described.

(a) *Inflamed glands.*—Inflamed femoral glands occupy the same position as a femoral hernia, and if the latter is irreducible the diagnosis may be difficult. With inflamed glands there are usually a shorter history, greater pain and tenderness, and the presence of more than one enlarged gland, with perhaps some evidence of the source of infection; whilst in the case of hernia it may be possible to feel the neck of the sac passing into the saphenous opening. If, however, the hernia has become inflamed, it may be impossible to distinguish between the two.

(b) *Lipoma.*—A lipoma due to a fatty overgrowth of the septum crurale is common, and is often associated with a small hernial sac. It is irreducible, and may have no impulse.

(c) *Varicose veins.*—A varicosity of the upper

end of the internal saphenous vein may resemble a femoral hernia, but is associated with varicosity of the veins of the limb, disappears when the patient lies down, and fills again even though the crural canal be occluded.

(d) *Psoas abscess.*—This is accompanied by a swelling in the corresponding iliac fossa, and other evidences of spinal disease are detectable.

UMBILICAL HERNIA.—The protrusion takes place at or about the umbilicus. Three main varieties are described: (1) *Congenital.* The normal closure of the abdominal wall is incomplete. All degrees may be seen. On the one hand, the greater part of the abdominal wall may be represented by amnion, the sac so formed containing many viscera; on the other, a small portion of intestine may alone be present in the base of the cord. (2) *Infantile.* This is due to yielding of the umbilical cicatrix and is very frequent; it tends to disappear spontaneously. (3) *Acquired.* Common in old, stout women, and rarely taking place at the umbilicus, this is really a mid-line hernia close to the umbilicus. It frequently contains omentum and large and small gut. The contents may become adherent and irreducible, and loculi may be formed. The hernia may increase to an enormous size, and the skin over the surface become infected and ulcerate.

Symptoms.—At the umbilicus a swelling is seen, which, in the congenital varieties, may be covered only by transparent amnion. It has a marked impulse, and is usually easily reduced. The acquired variety may be large and only reducible in part. When the abdominal wall is very fat the hernia may be overlooked because the sac has burrowed into the subcutaneous tissue. Since it contains large gut it is very prone to become obstructed.

VENTRAL HERNIA.—Here some portion of the anterior abdominal wall apart from the sites previously mentioned has yielded. There are two varieties, spontaneous and traumatic.

(1) *Spontaneous.*—These occur more commonly in the linea alba. They may be localized or diffuse. In the diffuse variety there is a separation of the recti, which may occur as a congenital lesion, as part of a general muscular weakness such as is seen in women with large families, or from a rapid increase in the intra-abdominal contents. The localized hernias occur in any position of the linea alba, and when present in the upper part are known as *epigastric hernias*. They are dependent on a small opening between the interlacing fibres of the linea alba. Through

HERNIA, ABDOMINAL

such an opening a small pad of extraperitoneal fat passes, bringing with it as it increases a pouch of peritoneum. They are more common in adults, are often very tender, and if containing omentum may give rise to marked gastric symptoms. Rarely, similar hernias may occur in the linea semilunaris.

(2) *Traumatic*.—These occur at the site of a scar following disease or operation. Sepsis or injury of a nerve with subsequent paralysis of a segment of muscle are the two most common causes of a yielding scar after operation. The hernia is generally gradual in onset. There is a progressively increasing bulge at the site of the scar, which is increased when the patient strains or coughs. These hernias are more common in the anterior wall, but may occasionally be seen in the lumbar region following operations upon the kidney. Owing to the width of the opening they do not often become strangulated.

OBTURATOR HERNIA.—In this case there is a protrusion along the course of the obturator vessels. The sac is small, and although a swelling has been described in the thigh it is very doubtful whether this ever occurs. As a rule the condition is only recognized owing to acute intestinal obstruction, and it is, in fact, one of the varieties of internal hernia. It is usually of the nature of a Richter's hernia.

DIAPHRAGMATIC HERNIA.—There is an opening in the diaphragm through which some of the viscera pass into the thorax. The opening is generally congenital, and is situated in the left posterior quadrant of the diaphragm. In rare cases it may be due to trauma.

Symptoms.—In many of the congenital cases there is dyspnoea, which is increased after the child is fed. In other cases very few symptoms may be present, so that the condition is overlooked until operation. In yet others the symptoms resemble those of some stomach lesion, because the gastric movements are interfered with. A positive diagnosis may at times be made by an X-ray photograph after a bismuth meal.

Treatment of hernia.—It is frequently said that young children affected with hernia should wear a truss, the theory being that if the intestinal contents be prevented from entering the sac the normal process of closure will take place. There is little or no evidence that this is so. In certain cases, if the sac is small, the viscera may not descend for many years. In one case, after a year of such treat-

ment no further symptoms presented themselves for eighteen years; but at operation a well-formed and widely-open sac was found. Operation should therefore be regarded as the treatment of choice. If the child is breast-fed this may be delayed until weaning is completed, the danger of strangulation in young infants being very slight. During the period of waiting, a woollen truss may be ordered so that the sac may be kept empty as far as possible. If the child is artificially fed, operation may be performed safely even within the first fortnight of life, provided that there is no contraindication. In infants such an operation is simple and the prognosis extremely satisfactory. If the child is weakly, if there are multiple hernias, or if the parents refuse operation, a truss may be worn. In quite young infants a woollen skein truss may suffice; but after the age of two or three months a small, accurately fitting rubber truss should be ordered. This should be worn constantly, being changed only while the patient is recumbent, as when he is being washed. Congenital umbilical hernias, if small, may be cured by the application of a pad, but, if large, can only be cured by operation, an attempt being made to repair the abdominal wall. Infantile umbilical hernias will in nearly all cases be cured by a pad, and operation should not be advised unless they are large.

For young adults with *inguinal* or *femoral* hernias a truss is often advocated. It is, however, uncomfortable to wear, has to be worn constantly, and will sooner or later fail in its purpose. For these reasons operation should always be the treatment of choice.

If there is some contraindication in the patient's general health—if he is aged, if the abdominal wall shows marked weakness, or if there be considerable increase in the intra-abdominal pressure—a truss may be ordered as a palliative measure.

Many forms of truss are now on the market, but the variety most commonly used is the circular spring truss. It must be made to fit well and be sufficiently strong to retain the hernia without causing discomfort, and must cover the neck of the sac. It is always better for the patient to be fitted by the instrument-maker, but if this is impossible it is only necessary to give the measurement round the body at a level passing over the base of the sacrum, midway between the crest of the ilium and the great trochanter, and well above the symphysis in front. Mention

HERPES SIMPLEX

must be made of the site and variety of the hernia, and whether a single or a double truss is required. For an acquired hernia a double truss should always be ordered. No truss should ever be applied to an irreducible hernia.

In *acquired hernias* the prognosis from operation is not so good. The abdominal wall is weak, and, since the neck of the sac is usually wide, especially in the inguinal varieties, the danger of strangulation is less—hence a truss may be advocated. If the hernia is large, painful, or insufficiently controlled by a truss, operation may be advocated.

Patients with *umbilical hernias* are, as a rule, unsatisfactory subjects for operation, but modern operations give good results. Unless the patient's general condition is bad, operation should be advocated owing to the high mortality associated with strangulation.

Diaphragmatic hernias should, if diagnosed, be operated upon, attempts being made to close the opening in the muscle.

In the first few hours of *strangulation* it may be permissible to attempt reduction by taxis, but it must be remembered that this method is associated with considerable risk, and, since any such hernia should be operated upon sooner or later, it is better that an operation be performed at once. Not only is there risk of injuring the gut or omentum by attempts at taxis, but the hernia may apparently be reduced and the symptoms still persist. The sac and its contents may be reduced *en masse*. The strangulation may be due not to the neck of the sac but to a band or hole in the mesentery within the sac, or even to a volvulus of the contained intestine. Again, gut may be already in part gangrenous when reduced into the abdominal cavity. For these reasons taxis should be attempted for only a short period and no undue force should be used. If it fail, operation should be performed at once.

A. J. WALTON.

HERPES SIMPLEX.—A common and troublesome complaint, characterized by the appearance of vesicles grouped on a tumid erythematous base. In certain cases a tendency to recurrence is a prominent feature.

Etiology.—No very definite information is available as to the cause of this eruption, but two factors seem to be associated with the disease sufficiently often to justify a belief in their causal relationship; these are trauma and infection. An instance of traumatic origin is seen in the appearance of the eruption

on the genitals after the sexual act; this may occur in either sex, in the female sometimes after the first act of intercourse. Among the infective types are those which appear during the course of some general disorder, such as pneumonia, meningitis, or a common cold. Recurrent herpes about the face may be related to oral or nasal sepsis.

Symptomatology.—Before the skin lesions appear, sensations of pain or burning are felt, followed by the rapid eruption of a cluster of vesicles, each about the size of a pin-head. In situations where warmth and moisture favour maceration, secondary infection frequently follows, in consequence of which there may ensue inflammatory changes of greater or less severity. The neighbouring glands may enlarge, or may even suppurate. In women a form is sometimes encountered involving the labia, and occasionally the pubic region also; there may be associated with this a mucopurulent discharge and severe pain. Although it sometimes assumes a more aggravated character in women than in men, genital herpes is less common in the former sex.

Diagnosis.—When the eruption is developed in its typical form, diagnosis is easy. Difficulty is most often experienced when infection or maceration has complicated or disguised the original disorder. On the face the disease may be confused with *impetigo*, from which it differs in that the essential lesions are in the form of grouped vesicles, while the "stuck-on" crusts of *impetigo* are wanting. The determination of the nature of a penile lesion is often far from easy, even for an experienced observer. The resemblance to a *chancre* is at times very close, especially if caustic or irritant substances have been used. If the case is one of herpes, careful inspection usually discloses a group of shallow, circular erosions, representing pre-existing vesicles, or a polycyclic arrangement, and examination for the *Spirochaeta pallidum* is negative. But the practitioner should nevertheless remember that both herpes and syphilitic infection may result from the same sexual act, and should warn the patient that subsequent development of the latter disease cannot be excluded. Herpes of the penis seems peculiarly prevalent among those suffering from gonorrhoea.

Treatment.—Only simple remedies should be used. It is sometimes possible to abort the eruption, when developing on the skin, by applications of absolute alcohol. After full development, bland powders like starch or talc

should be dusted on freely, or calamine lotion may be employed. Washing with boiled water or boric-acid solution forms a useful adjunct. For the severe type met with on the genitals of women, complete rest in bed and local application of calamine lotion are demanded. Septic infection when present calls for the usual methods. Most difficulty will be found in the treatment of the recurrent type of herpes; irradiation with X-rays is often curative, and should be tried when possible, especially for the eruption on the skin. If the situation permits of it, circumcision, by removing the area involved, serves to get rid of the recurring form met with on the prepuce. In some of these cases arsenic is most useful.

H. MACCORMAC.

HERPES ZOSTER (*syn.* Shingles; Zona).—An acute disease characterized by the appearance of erythematous patches, following a "root" distribution, upon which groups of vesicles appear.

Etiology and pathology.—The disease may occur at all ages and in both sexes. By some the eruption is regarded as a local manifestation of a general infection, the nature of which is unknown. The actual causal lesion is found in the posterior root ganglion, and this accounts for the peculiar distribution of the eruption. There appears to be a seasonal incidence, and epidemics have been met with. Some authorities maintain that the disease is allied to chickenpox, and instances of the simultaneous appearance of both diseases in the same household are cited as proof of this contention. It has long been held that persons taking arsenic are peculiarly prone to attack.

Symptomatology.—The first symptom often consists of neuralgic pain referred to one of Head's areas. Enlargement of the neighbouring lymphatic glands may accompany or even precede the eruption. In any case, tumid red areas soon appear along the selected site, rapidly becoming studded with numerous discrete vesicles. The eruption is usually unilateral, a feature of diagnostic importance. In rare instances the disease assumes a bilateral form. One attack almost invariably confers immunity. As in vesicular eruptions in general, secondary infection is not unusual, when the contents of the vesicles become purulent. Neuralgic pain of varying degree is a common accompaniment, and may assume a severe form, especially in elderly people, in whom it may persist for long periods. In some cases

the fifth nerve is implicated, one segment being usually selected. Where the eye is involved, especial care should be taken to protect it from further damage from outside agencies.

Diagnosis.—The condition may be mistaken for *intercostal neuralgia* or *pleurisy* before the eruption appears; when this is fully developed, diagnosis is usually easy. Occasionally the vesicles contain blood, constituting the so-called hæmorrhagic zona, or a form may be met with in which the lesions become gangrenous. When a patient presents himself with neuralgic pain following an attack, the characteristic scarring serves as a guide to the cause of the pain. In *herpes simplex* the construction of the individual patches is somewhat similar, but the segmental arrangement is wanting. The lower degree of temperature assists in distinguishing zona from *erysipelas*. *Eczema* has no segmental arrangement.

Treatment.—This resolves itself into prevention of secondary infection and relief of pain. It is often stated that simple painting with collodion is sufficient for the former purpose, but if pyococci have already obtained entrance this procedure may be attended by considerable suppuration. It is, therefore, not entirely free from criticism. A safe and useful plan, suitable for the great majority of cases, consists in washing the affected parts with 1-in-1,000 perchloride of mercury lotion, dusting with a powder composed of boric acid 1 dr., zinc oxide and powdered starch $\frac{1}{2}$ oz. each, and then covering with cotton-wool. When infection is present, appropriate antiseptic dressings are demanded. Quinine and iron tonics are useful in severe forms.

If relief from pain is required, aspirin or antipyrin usually proves sufficient; but difficulty will be experienced in treating cases in which severe neuralgia persists after the original attack has terminated. Here irradiation of the painful areas with X-rays proves of great benefit.

H. MACCORMAC.

HETEROCHROMIA (*see* EYE, CONGENITAL ANOMALIES OF).

HETEROPHORIA (*see* STRABISMUS).

HICCOUGH.—Irregular sudden contraction of the diaphragm, so that air is indrawn while the glottis remains closed, is responsible for the characteristic sound. Hicough is usually transient and of little importance, but may be a serious or even fatal complication of disease.

HILL DIARRHŒA

Etiology.—Repletion, flatulence, or the ingestion of hot or irritating food, such as peppery soups, are common causes. A hurried meal is sometimes responsible. More serious is the hiccough which occurs in general or local peritonitis, in acute intestinal obstruction, in acute colitis and dysentery, and in typhoid fever, for it materially exhausts the patient and banishes sleep. In these conditions it is of evil omen. A troublesome and painful form may be due to inflammation of the diaphragmatic pleura, either alone or in association with pneumonia. It sometimes occurs in pericarditis, in valvular disease, and in mediastinitis, and is then a serious complication. It is met with also in hepatic disease, in toxic states (especially uræmia), in conditions of profound exhaustion, and in association with organic nervous disease. A very stubborn form is seen in hysteria, but in this affection it rarely interferes with sleep.

Treatment.—Many well-known devices are used to allay the inconsequential form: sipping water, holding the breath, tickling the nostrils, compression of the diaphragm. If it is more intractable, a tumblerful of warm water containing sod. bicarb. (20 gr.) and peppermint is usually successful. For the severer and exhausting forms, sedatives are indicated. Bromides, chlorotone (15 gr.), or phenacetin (10 gr.) may be all that is required, but opiates or even chloroform anæsthesia may be necessary to give the patient some respite from his exertions and induce sleep. Apomorphine hypodermically ($\frac{1}{4}$ gr.) is sometimes strikingly successful. Peripheral stimulation by a blister on the epigastrium, compression of the vagus in the neck, or faradism may be tried, and a tight bandage or plaster around the upper part of the abdomen may give relief. These local measures, combined with suggestion, are the most satisfactory in hysterical cases.

FREDERICK LANGMEAD.

HIDROCYSTOMA (see SWEAT-GLANDS, AFFECTIONS OF).

HIDROSADENITIS (see SWEAT-GLANDS, AFFECTIONS OF).

HILL DIARRHŒA.—A form of diarrhœa affecting chiefly Europeans who sojourn in the hills after leaving the plains of tropical countries. Though long recognized and commonest in India, it occurs also in the highlands of Europe, South Africa, and South America. An altitude above 6,000 feet, combined with con-

siderable humidity, provides the climatic conditions under which it is prone to occur, and it is therefore commonest during the rainy season and is most prevalent in years with the greatest rainfall. To sprue it bears distinct resemblance; though ordinarily less severe, if protracted it may come to be distinguishable clinically from that disease only by the absence of the stomatitis.

Symptoms.—The chief feature is a looseness of the stools, which recurs daily in the early morning hours, and demands prompt evacuation of the bowels. The motions are white and frothy, and generally quite fluid, though they may be pasty. There is considerable flatulence, which causes great abdominal distension, and there is also discomfort from restless, churning peristaltic movements, but no serious pain. After the evacuation of the characteristic stools, which may be few or many, the diarrhœa ceases during the course of the morning, leaving the patient free to carry on his ordinary activities without hindrance, but returning again next day.

Prognosis.—The diarrhœa may subside even without treatment within a few days or weeks, or despite every care may continue until the patient returns to the plains. Sometimes its severity is such that the general health becomes deleteriously affected, anæmia, loss of weight, and general feebleness ensuing, and a state of chronic ill-health and diarrhœa being entered upon.

Treatment.—Crombie, who has carefully studied the disorder, recommends a diet of milk only, rest, warm clothing, and the administration of liq. hydrarg. perchlor. in drachm doses in water about fifteen minutes after food, followed by a digestive, such as pepsine 12 gr., two hours later. Return to the plains, the surest form of treatment, should not be postponed if such measures are unsuccessful.

FREDERICK LANGMEAD.

HILUS TUBERCULOSIS (see PULMONARY TUBERCULOSIS).

HIP-JOINT DISEASE (see ARTHRITIS, TUBERCULOUS).

HIRSCHSPRUNG'S DISEASE (see COLON, DILATATION OF).

HOBNAIL LIVER (see LIVER, CIRRHOSIS OF).

HODGKIN'S DISEASE (see LYMPHADENOMA).

HORDEOLUM (see EYELIDS, AFFECTIONS OF).

HYDATID DISEASE

HORMONES, TREATMENT BY (see ORGANOTHERAPY).

HORSESHOE KIDNEY (see KIDNEY, CONGENITAL ANOMALIES OF).

HOURL-GLASS STOMACH, SPASMODIC (see STOMACH, FUNCTIONAL DISORDERS OF).

HOUSEMAID'S KNEE (see BURSAL ENLARGEMENTS).

HUNTINGTON'S CHOREA (see CHOREA, HUNTINGTON'S).

HYDATID DISEASE.—Hydatids are the larval or bladder-worm stage of the tapeworm, *Tania echinococcus*.

Etiology.—The adult tapeworm inhabits the small intestine of the dog, dingo, fox, and jackal, and there are usually a large number in each infected animal. It has four segments (Fig. 41), with a total length of less than 5 mm. The head has a rostellum of 30 to 40 hooklets and 4 sucking discs, and behind this there is a narrow neck. The second and third segments or proglottides are small; the fourth contains the mature sexual apparatus, and

exceeds the whole of the rest of the worm in size. There are about 500 ova, each of which has a 6-spined embryo enclosed in a thick chitinous shell. The ripe segments break off and, soon decaying, free the ova. These are ingested by domesticated animals, usually by sheep, more rarely by oxen and pigs. Man is generally infected by contaminated drinking-water or vegetables, less often directly from the coat of a pet dog.

On reaching the intestine the chitinous egg-shell is absorbed and the embryo released. It enters the portal circulation and most often comes to rest in the liver, but may settle down in the lungs, peritoneum, kidneys, brain, spleen, bones, or elsewhere. It is probably not entirely passive, but has some power of choosing a site for further development.

The frequency of the disease in a country depends on the readiness with which infection from the primary to the secondary and from the secondary to the primary hosts can be effected, and on the degree to which the habits of the people subject them to the risk of ingesting ova. It is in Australia and Iceland that the conditions for transmission are most perfect and that hydatid disease is consequently most common. It occurs much more frequently on the Continent than in England. In India and the tropics it is rare. Although many of the cases met with in this country are infected abroad, the disease is indigenous both in the large towns, including London, and in the country districts. It is commoner in adults than in children, and, in the British Isles, in males than in females.

Pathology.—When the embryo reaches its final resting-place it loses its spines and begins to grow very slowly, at first remaining solid and later acquiring a liquid central portion.

The organism consists of an outer stratified cuticle of chitin, the ectocyst, and an inner parenchymatous layer which contains muscle and a vascular system. Expansion takes place by the gradual addition of fresh layers of chitin to the ectocyst, and by the increase of fluid contents. Occasionally the hydatid remains in this condition, and is known as a sterile hydatid or acephalocyst. More usually, secondary vesicles arise inside the primary one, and even tertiary ones inside the secondary, and these are known as daughter and grand-

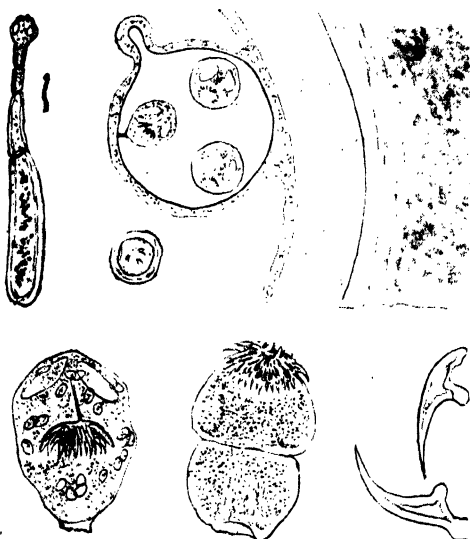


Fig. 41.—Diagram showing *Tania echinococcus* (natural size and enlarged), an ovum, section of wall of hydatid cyst with brood-capsule and scoleces, two scoleces, and two hooklets.

HYDATID DISEASE

daughter cysts. This is the common mode of development in man, but sometimes the secondary vesicles grow outwards from the primary or mother cyst, and either form a great aggregation of small cysts or wander along the fascial planes of the host, sometimes to a considerable distance. Cysts formed in this way do not occur in viscera, and are known as exogenous hydatids. The so-called alveolar form of hydatid, which occurs in man and the ox, and is confined to certain parts of Central Europe, is almost certainly the larval form of a distinct species of tapeworm.

From the parenchymatous layer of any of these cysts, primary, secondary, or tertiary, minute outgrowths appear, the brood-capsules (Fig. 41). When fully formed they are about 1.5 mm. in diameter and consist of two cellular layers which enclose a fluid centre. From the exterior of each brood-capsule a number of scoleces or heads of future echinococci develop as hollow buds. At the bottom of the hollow bud farthest from the brood-capsule, suckers and a row of hooklets are formed. Soon the scolex can invert itself, and it then projects into the interior of the brood-capsule at the end of a narrow stalk, and resembles closely the head of the adult worm. At this stage the brood-capsule frequently bursts, and the scoleces either become free or remain attached to the stalk of the brood-capsule. They are about 0.18 mm. in diameter. Several thousand brood-capsules may develop in one hydatid, and each may produce from 10 to 20 scoleces.

In the liver the irritation of the cyst causes a growth of fibrous tissue such as is produced by any foreign body, but the gradual increase of pressure causes atrophy, and so maintains the capsule at a more or less even thickness. This outer capsule formed by the host is best developed in the liver, and is almost absent in the brain.

More than one hydatid may be found in a single individual, and each of them is developed from a separate embryo. They may be due to more than one infection.

The cysts may live for many years in this condition, enclosing living scoleces, and, if the flesh of animals containing them is eaten by a dog, the scoleces cling by their suckers and hooklets to the mucous membrane of the duodenum and develop into adult worms.

In other cases the cyst may die from old age, trauma, or the introduction of some poison. For instance, the transudation of bile into a cyst invariably causes death, and

tapping frequently effects the same result. Calcification of the fibrous capsule, which often occurs, may exert a deleterious influence on the enclosed parasite. The first change takes place in the fluid, which becomes turbid and albuminous; the contents then become gelatinous from the precipitation of proteins, and cholesterol is often present. The mother cyst is affected first, but the daughter cysts eventually go through the same stages. Fatty changes take place and water is abstracted, so that the whole of the contents become transformed into a pultaceous mass, which contains crystals of cholesterol and stearin. The chitinous ectocyst may be thrown into folds, but always retains its distinctive characters; hooklets can be found in nearly every case by microscopic examination of the contents. By the infiltration of calcium carbonate and phosphate the mass may become finally of stony hardness.

Suppuration of a cyst is not a very uncommon occurrence, and may be spontaneous or may follow aspiration or other injury.

Symptomatology.—The growth of a cyst is so slow that in many instances no symptoms are produced, and a considerable number of hydatids are discovered post mortem in cases in which no suspicion of their presence had been aroused during life. Where symptoms are produced they vary much according to the organ affected. Where there is no room for expansion, as in the brain, symptoms occur early.

Hydatids of the brain.—These give rise to symptoms and signs due to pressure, and differ in no way from those caused by any slow-growing intracranial tumour. Headache, vomiting, fits, paralyses, and optic neuritis are the most prominent symptoms. The cyst or cysts may be situated between the brain and skull or in any part of the brain substance, and at any depth, sometimes even in one of the ventricles. They are much commoner in the cerebrum than in the cerebellum. Localizing symptoms may or may not be present. Local bulging of the skull, sometimes accompanied by marked thinning of the bone, may be produced, and usually indicates that the cyst is extracerebral.

Hydatids of the spinal cord are very rare and produce the symptoms of tumour.

Hydatids of the lung.—The symptoms manifested depend largely on the size of the cyst. Cough is almost invariably present and may be severe. Hæmoptysis is also a very common

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companionment, but usually consists merely the expectoration of blood-streaked sputum, and is caused by passive congestion of the bronchi due to pressure. Dyspnoea is generally attendant with small hydatids, and seldom pronounced even with large ones. Pain is not a prominent symptom in the absence of suppuration, though an uncomfortable sense of weight may be noticed. Pyrexia and wasting are absent, unless pleurisy or infection of the cyst has occurred. The physical signs vary both with size and with situation. There may be local bulging of the chest-wall, and part of the bone of one or more ribs may be absorbed. In such cases there may be an impulse on coughing. A superficially situated cyst gives rise to marked dullness and a sense of great resistance on percussion.

Skodaic resonance may be present above the cyst owing to relaxation of the lung, and has been noticed even over the site of the cyst itself. Breath-sounds are weak or absent, and the vocal vibrations and vocal resonance are reduced. The heart is frequently displaced. Rupture into a bronchus is not uncommon, and the sudden expectoration of watery fluid and of colourless hydatid membranes makes recognition easy.

Hydatids of the liver.—On several occasions I have met with hydatids of this organ which gave rise to no symptoms during the patient's life. If very large they cause a sense of fullness in the hepatic region and aching near the right shoulder-blade. Small hydatids or moderately large ones lying deep in the liver substance cause no physical signs. Large ones in the centre of the organ cause a general enlargement of the lobe in which they are situated, usually the right, but the surface of the liver remains smooth. A cyst in the upper part may be difficult or impossible to distinguish from one in the lower lobe of the lung. Lung and heart may be very much displaced; there may be bulging of intercostal spaces, a dome-shaped area of intense dullness, and weak or absent breath-sounds. There is, however, no band of resonance or lessened dullness between this area and the upper edge of the liver, and the liver itself is usually displaced farther downwards than in primary hydatid of the lung.

In hydatid disease near the lower border of the liver one or more rounded swellings can generally be felt. They are smooth and elastic, and in some cases become pedunculated. The hydatid thrill, obtained by placing the

middle finger over the centre of the tumour while the first and third fingers are spread over the outer portions, and percussing it sharply, is seldom felt, nor is it pathognomonic of this condition. Jaundice is rarely present in uncomplicated hydatid disease, though biliary colic with jaundice is occasionally produced by rupture of the cyst into a bile-duct. Besides rupture into a bile-duct, rupture into the stomach, intestine, bronchus, pleura, peritoneum, or elsewhere occasionally happens. Empyema or pyopneumothorax generally follows rupture into the pleura, and a fatal peritonitis that into the peritoneum.

There may be merely a subacute peritonitis with ascites, sometimes followed, after a long interval, by multiple hydatids of the peritoneum, most abundant in the omentum and pelvis.

Hydatids of the spleen may give rise to no symptoms, or to a sense of weight. There may be a smooth swelling with the ordinary characters of an enlarged spleen, or with multiple cysts the swelling may be nodular. Occasionally the cyst becomes pedunculated and freely movable.

Hydatids of the kidney.—A sense of weight and discomfort in the lumbar region may be produced by a large cyst. Rupture is a common sequel, and the first symptoms noticed may be due to this event. Severe renal colic is often caused. The physical signs are those of renal tumour. A somewhat movable tumour, which can be pressed up by a hand placed on the loin behind, and can be separated on deep inspiration from the lower edge of the liver or the spleen, is found. The tumour may be smooth and globular or nodular, according to the presence of one or more cysts. There is usually a band of resonance between the upper part of the swelling and the lower edge of the liver when the right kidney is affected.

Hydatids of the omentum and peritoneum are usually multiple, and may be freely movable or fixed. Sometimes they are very hard owing to the thickness of their capsules.

Hydatids of bone may cause a large tumour with thinning of bone so as to produce egg-shell crackling.

Hydatids of muscle are rare; painless cystic swellings are found.

Diagnosis.—A rounded painless tumour, firm and smooth, which has given rise to no special symptoms and little or no disturbance of the general health, is likely to prove to be a hydatid cyst. The presence of a hydatid thrill would confirm the diagnosis. A history that

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the patient had lived in or visited one of the countries where the disease is very prevalent, such as Australia, would be additional evidence. Proof of the existence of previous hydatids removed by operation or coughed up would be very helpful. Aspiration may be carried out, and a small quantity of fluid obtained in this way can be tested. Clear fluid of low specific gravity and nearly neutral reaction, and containing little or no albumin and a large amount of sodium chloride, is probably derived from a hydatid cyst. Unfortunately this simple method is by no means free from danger, and in Australia is practised much less frequently now than formerly.

Eosinophilia is frequently present, but not universally so. Its absence does not indicate absence of the parasite, and its presence may be due to any of the larger helminths. It is of far greater significance in people who have never left England than in those who have lived abroad where parasitic worms are so common.

Precipitin test.—This test is dependent on the fact that if a few drops of serum of a patient with hydatid disease are added to hydatid fluid a precipitate is formed in 18–20 hours, whereas the serum of a healthy person gives no precipitate. Positive results are obtained in a large percentage of all cases even when suppuration of the cyst has taken place.

Bordet-Gengou reaction.—This test depends on the fixation of complement, and the technique is complicated. Properly carried out, a large proportion of cases give positive results. This reaction, like the precipitation test, will be of little practical value until some method of preserving hydatid fluid for an indefinite period has been devised. At present it can only be kept for about three months.

X-rays.—Skiagrams are of great value, especially in intrathoracic parasites. The circular outline of the shadow in many cases makes the diagnosis certain (see PLATE 45, Fig. 3, Vol. III, facing p. 556). They may be of some use in bone disease.

The foregoing methods of diagnosis apply to all cases of hydatid infection.

The most important points in the differential diagnosis of echinococcus disease of special organs from other diseases which may affect them similarly will now be considered.

Brain.—Hydatid disease in this organ occurs in early adult life in the majority of cases. It is rarely recognized before operation, in the absence of evidence of present or previous

hydatid disease. Cerebellar tumours in a child are practically never hydatids, and in an adult very rarely.

Lung.—Here the disease must be differentiated from—

(1) *Pulmonary tuberculosis.*—When the signs are sufficiently marked to resemble those of hydatid cyst there will be evidence of disease of the opposite side in most cases. Bilateral hydatids are rare. Tuberculosis is generally apical, hydatids are basal.

(2) *Pleural effusion.*—Marked convexity of the upper limit of dullness at the base behind suggests hydatid; the convexity is in the axilla in effusion. Resonance at the extreme base excludes effusion. Greater displacement of the heart than would be expected from the extent of dullness is in favour of hydatid cyst.

(3) *Aneurysm.*—If a swelling projects from the chest-wall, pulsation, if present, will not be expansile in hydatid. A negative Wassermann reaction is against aneurysm. Screen examination may show whether the tumour pulsates or is in connexion with the aorta, if the aneurysm or cyst is deep-seated.

Liver.—Hydatids must be differentiated from the following conditions which give rise to general enlargement of the liver:—

(1) *Nutmeg liver.*—This is tender, and the signs and symptoms of morbus cordis are prominent.

(2) *Amyloid disease.*—Other organs are generally affected. Albuminuria and diarrhoea are frequently met with. The general health is poor. Evidence of syphilis, pulmonary tuberculosis, or longstanding suppuration is obtainable.

(3) *Leukamia.*—The general condition and the blood-count should prevent error. The spleen is usually greatly enlarged.

(4) *Lymphadenoma.*—There is severe anæmia and frequently pyrexia, with enlargement of lymphatic glands and spleen.

(5) *Malignant disease* is rarely primary, and when secondary a primary growth can be found in more than half the cases. Pain, rapid increase in size of liver, and jaundice are common in this condition, rare in hydatids. Loss of health is pronounced.

(6) *Fatty cirrhotic form of portal cirrhosis.*—Though usually smooth, the liver in this condition is very hard. There is often evidence of portal obstruction—dilated veins round the umbilicus, piles, hæmatemesis, or ascites. Digestive disturbances and a history of alcoholism, especially of beer-drinking, may be obtained.

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(7) *Hepatoptosis*.—The liver is displaced downwards, but not increased in size as in hydatid disease.

Hydatids causing single or multiple local enlargements of the liver must be diagnosed from—

(1) *Enlarged gall-bladder*.—This is more movable, and there may be a history of attacks of biliary colic or jaundice.

(2) *Cholecystitis*.—Adhesions may prevent movement, but pain and tenderness are too great to cause confusion with a non-suppurating hydatid.

(3) *Simple cysts* are seldom large enough to be felt, and will be accompanied by bilateral tumours due to cystic kidneys in the flanks.

(4) *Tropical abscess* may be indistinguishable clinically from suppurating hydatid. It is only met with in those who have been exposed to infection by the *Entamoeba histolytica* in the tropics, though it may not develop until years afterwards. A history of dysentery can generally be elicited.

(5) *Malignant disease*.—There may be one large or many smaller nodules, but they are harder than hydatids and often umbilicated. Other points of difference have already been mentioned.

(6) *Syphilitic disease*.—A hard, irregular surface is produced, and there is often ascites. Anæmia, other syphilitic lesions, and a positive Wassermann reaction will support the diagnosis.

(7) *Hydronephrosis*.—In this condition the hand can usually be pushed between its upper border and the lower edge of the liver, but not in the case of hydatid. There may be a history of the passage of much urine of low specific gravity, whereas this cannot occur in hydatid disease unless the cyst has ruptured into the pelvis of the kidney.

Hydatids have to be differentiated also from pancreatic, omental, and ovarian cysts, as well as from ascites. *Pancreatic cyst* is more central than hydatid, and lies behind the stomach, which can be inflated to prove this. *Omental cysts* are more mobile. *Ovarian cysts* can generally be traced to their pelvic origin. In *ascites* the flanks are dull; in the largest hydatid they remain resonant.

Hydatids of the kidney are nearly always thought to be hydronephrosis unless the cyst ruptures and membranes are passed.

Hydatids of the spleen are rarely recognized unless the hydatid thrill is present.

Hydatids of the omentum and mesentery are

indistinguishable from pancreatic, mesenteric, or even ovarian cysts.

Peritoneal hydatids are generally pelvic, and usually mistaken for ovarian tumours or sub-peritoneal fibroids.

Hydatids of bone are usually mistaken for endosteal sarcomata.

In diagnosis it is important to remember the relative frequency of the occurrence of hydatids in the various organs. Thomas gives the percentages as—liver 57, lungs 11.6, kidneys 4.7, brain 4.4, spleen 2.1, heart 1.8, peritoneum 1.4.

Prognosis.—This depends largely on the situation of the tumour, and on the absence of suppuration. Hydatids of the brain are nearly always fatal. Recovery from hydatids of the lung is not infrequent, and may take place even after rupture spontaneously or following operative interference, though the mortality after rupture is about 50 per cent. Death may be caused by hæmorrhage from erosion of a large vessel or rupture of a small aneurysm.

Secondary infection of a cavity may cause intermittent fever, wasting, and death from exhaustion. Empyema and pyopneumothorax are very fatal complications, causing death in 80 per cent. or more of cases.

Hydatids of the abdominal viscera are always serious, but are often completely cured by operation. Owing to the possibility of other and deeper-seated cysts, the prognosis is always doubtful.

Rupture of a cyst into the peritoneum causes a mortality of 90 per cent.

Treatment.—Two methods of treatment are commonly adopted—(1) aspiration, (2) incision followed by removal of the cyst.

Aspiration of a small quantity of fluid may kill the parasite and lead to the degenerative changes previously described, but it fails in many cases entirely, or only the mother cyst dies. In any case the cyst remains a permanent source of danger. Simple as the procedure is, it is not free from risk. The fluid contents may leak into a vessel or into the peritoneum, and if rapid absorption takes place there may be severe collapse with diarrhoea and vomiting, or even death. The escape of a small quantity may cause unpleasant symptoms, including urticaria. If fluid leaks into the peritoneum it may cause peritonitis with ascites, and later on infection with multiple hydatids. Aspiration may also cause suppuration of the cyst, making operation imperative

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and much more dangerous. In the case of the lung there have been numerous instances of sudden death owing to rupture of the cyst and consequent flooding of the lungs with its contents, and suffocation.

In Australia aspiration as a means of diagnosis and treatment has been largely superseded by exploratory incision and removal of the entire cyst. Aspiration of a pulmonary hydatid is never justifiable except in an operating theatre after excision of a portion of rib.

If aspiration is undertaken the dangers are reduced by using a needle of small calibre.

A suppurating hydatid must be treated as an abscess by incision and drainage.

Hydatids of bone usually require amputation ultimately, though occasionally local removal is possible.

E. A. COCKAYNE.

HYDRAMNION. — Hydrannion or polyhydrannion is the name given to the formation of an excessive amount of amniotic fluid. The liquor amnii appears at a very early stage of development of the embryo, and gradually increases in amount up to about the seventh month of pregnancy, when it is relatively abundant compared to the size of the fœtus; after this date its production appears to be more or less stationary until, at term, it little more than fills in the interstices between the irregular fetal mass and the uterine wall. Its amount at term varies widely within normal limits, and averages about two pints; any amount less than half a pint (oligohydrannion) and more than four pints (polyhydrannion or hydramnion) is regarded as pathological.

The liquor amnii is a fluid of low specific gravity containing 90 per cent. of water, a small percentage of urea and inorganic salts, and a trace of albumin; it is usually clear, but is often turbid from mixture with vernix caseosa and fetal epithelium. As regards the source of the liquor amnii there is still some difference of opinion; it is probably mainly a secretion from the epithelium lining the amnion. There is little doubt that the fœtus also makes a contribution by excreting urine into it—the constant presence of urea in the liquor amnii is strongly in support of this. In cases of acute hydramnion associated with uniovular twins—to be referred to shortly—there can be no doubt that the enormous accumulation of liquor amnii is due to fetal polyuria.

Etiology.—Most cases of hydramnion are unaccompanied by any obvious cause. Very seldom is a maternal cause apparent; condi-

tions associated with generalized oedema—such as cardiac, renal, and hepatic disease—scarcely ever bring about an increase in the amount of liquor amnii. On the other hand, ovular or fetal abnormalities are fairly often associated with hydramnion. These are: (1) Malformation of the fœtus, such as anencephaly, spina bifida, ectopia vesicæ, and hare-lip. (2) In uniovular or homologous twins acute hydramnion is not uncommon, the amniotic sac of the stronger twin being the one affected; the stronger twin gradually appropriates more than its fair share of fetal blood, through the anastomosing umbilical vessels of the common placenta; its heart and kidneys become enormously hypertrophied, and fetal polyuria is the source of the excess of fluid.

In cases of hydramnion the fluid shows no abnormal characteristics. It is probable that only amounts of over four pints cause clinical features recognizable as hydramnion; cases with as much as thirty or forty pints have been recorded, but are exceedingly rare.

Symptomatology.—The physical signs are easy to appreciate: the uterus is larger than it should be for the period of pregnancy reached, and it feels like a thin-walled cyst through which a pronounced fluid thrill can be obtained in all directions; if there is much fluid the fœtus can be neither felt nor heard. The symptoms are caused by the pressure of the enlarged uterus, and consist chiefly of embarrassment of respiration and cardiac action varying with the degree of enlargement. There may be oedema of the legs, and there is not uncommonly slight albuminuria.

Two clinical forms are recognizable, the acute and the chronic. The acute form is rare; it usually begins at the fourth or fifth month, and enormous quantities of fluid quickly accumulate. The uterus rapidly attains a great size and pressure symptoms are correspondingly severe; there are abdominal discomfort and pain, dyspnoea, oedema of the legs, and often albumin in the urine. Such cases usually end in abortion at about the sixth or seventh month, heralded by rupture of the membranes, which brings instant relief. Cases of acute hydramnion are seldom found apart from uniovular twins.

In the chronic form, much the commoner, the distension is more gradual, and the symptoms are correspondingly less severe. The uterus may gradually reach an enormous size, though the slighter forms are far more usual.

Diagnosis.—It has already been stated that

only amounts of more than four pints are recognizable clinically as cases of hydramnion. It is often difficult to distinguish between hydramnion and *twin-pregnancy*, and the two are often associated. There should be no difficulty in distinguishing hydramnion in the early months of pregnancy from a case of *vesicular mole*; in the latter the uterine enlargement is not nearly so rapid and there is usually irregular uterine hæmorrhage. Two conditions which may sometimes confuse are ovarian cyst and ascites. The presence of the presumptive signs of pregnancy and careful physical examination of the abdomen, especially of the distribution of the areas of dullness and resonance, ought always to make the diagnosis between hydramnion and *ascites* an easy matter. The diagnosis between hydramnion and *ovarian cyst* will, in doubtful cases, always be made clear by resorting to an examination under anæsthesia; in the latter the small uterus will be felt distinct from the main tumour. The diagnosis between hydramnion and pregnancy associated with a large thin-walled ovarian cyst is often difficult: the points to be sought for, in the latter, are the recognition of two distinct tumours, differing in their physical characters—one of the tumours will give the physical characters of the pregnant uterus, i.e. the elastic consistency, the contractions and relaxations, the feeling of foetal parts and the hearing of foetal sounds; the presumptive signs of pregnancy accompany both conditions, of course.

Treatment.—It is impossible to control the production and absorption of the liquor amni; therefore, therapeutic treatment is of no use; the action of diuretics, diaphoretics, and cathartics has no effect in diminishing the quantity of the fluid. In many cases, relief is brought about by spontaneous rupture of the membranes followed by abortion or premature labour; this is especially likely to occur in the acute form. If the pressure symptoms become intolerable, abortion or premature labour must be induced, and this is best done by rupturing the membranes with a sound (this little operation must be performed with strict asepsis). Special precautions should be ready to deal with post-partum hæmorrhage in these cases, as efficient retraction of the uterus is apt to be delayed by the over-distension. The accoucheur should also be on the alert for foetal dangers: transverse and other malpositions are common, and the umbilical cord may be swept through the cervix by the gush of fluid.

In cases where the accumulation of fluid is not great enough to call for the interruption of pregnancy, the patient must be made as comfortable as possible by rest and posture. She will have to spend most of the day at rest, and will be more comfortable and less dyspnoic when propped up in a sitting position than when lying down.

EARDLEY HOLLAND.

HYDRARTHROSIS, INTERMITTENT.—A condition in which at regular intervals a recurrent effusion into a joint occurs.

The **etiology** is obscure, but the first attack usually follows a slight injury. The affection is commoner in women, and begins before middle age; sometimes the effusions correspond with menstrual periods, and are absent during pregnancy.

Symptoms.—At almost exact intervals, which differ in different cases, but are usually about ten days, the affected joint suddenly fills with fluid, which, as a rule, lasts less than a week. Between the attacks the joint may appear normal, or there may be slight thickening of the retropatellar pad of fat, laxity of ligaments, or signs of chronic toxic arthritis.

Prognosis.—The condition generally lasts for years, but it may disappear for no apparent reason, especially during pregnancy.

Treatment.—Foci of chronic infection, especially in teeth and gums, must be looked for and treated, and any local defects of the joint must be attended to. Treatment has little influence in cases where no local or general cause for the condition is found. Improvement has in some cases followed the use of arsenic, quinine, or mercury. As local treatment, massage, radiant heat, electricity and ionic medication are used; and aspiration of the effusion, followed by injection of iodine, may do good.

C. W. GORDON BRYAN.

HYDROCELE.—A serous effusion into the tunica vaginalis (Vaginal Hydrocele), or into a cavity derived from an unobliterated processus vaginalis (Hydrocele of the Cord; Hydrocele of a Hernial Sac).

Etiology.—Hydrocele may occur at any age, and in children the sac may communicate with the peritoneal cavity. It may be secondary to, and possibly mask, some inflammatory disease of the testicle, either acute or chronic. Ordinary vaginal hydrocele, though apparently sometimes due to injury, or some interference with the circulation, is generally idiopathic.

HYDROCELE

The sac, originally thin and supple, may, in old-standing cases, become thick and hard. The fluid is usually straw-coloured, but may become dark brown and contain cholesterol. The testicle, unless adhesions are present, will be behind and below.

Symptoms.—Hydrocele forms a smooth, tense, painless, elastic, scrotal or inguino-scrotal swelling not adherent to the skin and concealing the testicle. Except in old-standing cases the swelling is translucent, and, if grasped firmly by one hand and lightly percussed, gives a characteristic thrill.

Diagnosis.—Though diagnosis is generally easy, hydrocele may have to be distinguished from a hernia, which in children may also be translucent, from hæmatocele, and occasionally from a growth of the testicle.

Treatment.—Essentially a palliative measure, *tapping* is especially indicated in elderly men. The patient being seated, the surgeon, after verifying the position of the testicle and having sterilized the skin, grasps the hydrocele with his left hand and plunges the trocar and cannula into the swelling at a spot free from veins just below the middle of the anterior surface, tilting the point upwards as soon as the wall is penetrated. When the sac has been emptied the cannula is withdrawn and a sealed dressing applied.

Injection.—This method attempts to produce a cure by exciting plastic inflammation leading to the obliteration of the cavity. The sac having been emptied as described above, either 2 dr. of tinct. iodi fort., or $\frac{1}{2}$ –1 dr. of a strong solution of carbolic-acid crystals in glycerin, is injected through the cannula. The patient should be kept in bed for forty-eight hours. The method is not without danger, as sloughing and suppuration may occur, and it should never be employed in children.

Excision of the sac.—The hydrocele is exposed by an incision in the front of the scrotum. After it has been freed from the surrounding tissues and extruded through the wound it is widely opened and the whole of the parietal layer cut away, the visceral layer being treated with iodine. Absolute hæmostasis is essential, and drainage is necessary.

Inversion of the sac.—The hydrocele having been exposed, an incision about an inch long is made at its upper end. The testicle is drawn through this, and the sac is turned inside out so that the serous layer is in contact with the scrotal tissues. Two or three catgut

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stitches are passed through the divided edges of the tunica vaginalis, now at the back of the testicle, in order to maintain the inversion.

The results of either of the last two operations are usually satisfactory, though a recurrence occasionally takes place.

PHILIP TURNER.

HYDROCELE OF CANAL OF NUOK (see VULVA, DISEASES OF).

HYDROCEPHALUS.—This condition is due to an accumulation of fluid within the skull, either in the subdural space—external hydrocephalus—or inside the ventricles, distending the brain—internal hydrocephalus. The external form, which is very rare, occurs sometimes as a congenital abnormality in association with a small undeveloped brain. The internal variety is the more common, and is what is usually meant when the term hydrocephalus is used. It may be either congenital or acquired.

CONGENITAL HYDROCEPHALUS

Etiology and symptoms.—Congenital, idiopathic, or essential hydrocephalus shows itself at birth or in early infantile life. Its cause is obscure, but it has been ascribed to stasis of the ventricular fluid owing to displacement or obliteration of the foramina of communication of the ventricles with one another or with the subarachnoid space, as the result of congenital malformations or prenatal inflammatory conditions involving the ependyma and meninges. In some cases there seems to be a family disposition to the affection. It may be due also to foetal syphilis.

As the fluid distends the ventricles the surrounding brain substance becomes thinned and atrophied. In severe cases it may form only a narrow wall with flattened and poorly developed convolutions on its surface, while the fontanelles are widened and tense, the bones of the skull separated, and the head enlarged. The size of the head is the most characteristic feature of the condition and is in conspicuous contrast with the small face. The head is usually rounded or dolichocephalic and bulges in all directions; the forehead is prominent and overhanging, and the eyeballs are directed downwards and laterally.

The child is apt to be feeble and poorly nourished, and shows various symptoms dependent on the defective development of the brain. There may be blindness from atrophy

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of the optic nerves due to pressure on the chiasmal region. Sometimes convulsions occur, and not infrequently the limbs are spastic and weak, resembling cerebral diplegia, especially in that the legs are more affected than the arms. Later, mental symptoms show themselves in various degrees of backwardness and lack of development beyond the infantile stage, even to extreme idiocy, although in some cases the mental symptoms may be surprisingly slight.

The **diagnosis** is usually easy; if the child survives, the condition may be confused with *ricketts*. Death frequently occurs from wasting and exhaustion early in infancy.

Treatment is unsatisfactory. In severe cases the excess of fluid may be removed by ventricular puncture, or sometimes by lumbar puncture, and the skull compressed by means of rubber bandages; but compression is not without danger, and the fluid usually accumulates again quickly after removal. Various operations have been devised to establish a permanent drainage, but the results have not been encouraging. In some cases the administration of mercury and potassium iodide may be beneficial.

ACQUIRED HYDROCEPHALUS

Etiology.—The acquired form occurs in children or adults, usually secondary to and in association with meningitis or cerebral tumours. Meningitis leads to thickenings and adhesions which block the foramina of communication between the ventricles and the subarachnoid space, especially the foramina of Magendie and Luschka, with consequent damming up of the fluid in the ventricles. The secretion of cerebro-spinal fluid is probably also increased as a result of the inflammation. The two forms of meningitis with which hydrocephalus is particularly associated are posterior basic meningitis in infants and cerebro-spinal meningitis in older children and adults.

Cerebral tumours give rise to the condition by causing mechanical obstruction in the ventricular system. In the neighbourhood of the pons or cerebellum they may block the fourth ventricle or the aqueduct; near the base of the brain or in the midbrain may occlude the iter or the third ventricle, or press on the veins of Galen. The ventricular distension resulting from tumours is often slight and localized.

In infants or young children acquired hydrocephalus leads to an enlargement of the skull

and a condition similar to that found in the congenital form of the disease. In older patients expansion of the head is usually impossible, but occasionally the sutures separate, and not infrequently there is considerable thinning of the bones.

The **symptoms** are difficult to differentiate from those of the primary disease, as the most important are headache, drowsiness, mental impairment, optic neuritis or atrophy, attacks of rigidity or convulsions, and cerebellar symptoms such as ataxia or hypotonia of the limbs. The persistence or recurrence of some of these symptoms after the primary condition has subsided, or sudden exacerbations of them during its course, are suggestive of hydrocephalus, but the diagnosis in the absence of enlargement of the skull is uncertain.

In acquired hydrocephalus of any severity the **prognosis** is bad, but some cases come to a standstill. The **treatment** is involved in that of the primary condition to which the hydrocephalus is due. Acute distension of the ventricles, such as occurs occasionally during cerebro-spinal fever and in other conditions, may be relieved by lumbar or ventricular puncture.

MENINGITIS SEROSA, or acquired idiopathic hydrocephalus, is a rare condition in which an effusion of serous cerebro-spinal fluid takes place into the ventricles owing to excessive secretion of the ependyma or choroid plexus. It has been supposed to be inflammatory in origin, but so far no organisms have been found associated with it. The fluid is usually clear, of low specific gravity, and normal in character, except that it is under increased pressure.

The condition occurs in adults and in children, in either an acute or a subacute form. The symptoms of the acute type are similar to those of ordinary meningitis, while the more subacute cases simulate cerebral tumours. The diagnosis from these two conditions is very difficult and sometimes impossible.

The patients may recover completely, or a thickening of the ependyma may supervene which leads to a more chronic condition. The most rational treatment is removal of the excessive fluid by lumbar puncture. Mercurial treatment is also said to be of value.

P. W. SAUNDERS.

**HYDROCHLORIC ACID, POISONING
BY (see POISONS and POISONING).**

HYDRONEPHROSIS

HYDROOYANIC ACID, POISONING BY (see POISONS AND POISONING).

HYDRONEPHROSIS.—Distension of the renal pelvis with urine due to an increase of the urinary pressure behind some obstruction. If the obstruction is situated in the urethra, both kidneys and ureters are dilated; if in the course of one ureter, the corresponding kidney alone is affected. Occasionally a partial hydronephrosis is caused by the blocking of one or more calyces.

Etiology.—In true *congenital* hydronephrosis, which is usually bilateral, the kidneys are dilated at birth, and the infant rarely survives. Hydronephrosis dependent upon some other congenital abnormality may not become apparent till later. These cases are bilateral when they are due to phimosis, or congenital urethral stricture; unilateral when due to congenital stricture, valves, or kinks in the ureter, or at the uretero-pelvic junction, or to pressure on the ureter as by abnormal renal vessels.

Acquired hydronephrosis may be bilateral, from stricture, from enlarged prostate, from pressure on or involvement of both ureters by a new growth, e.g. a myomatous uterus or a cancer of the bladder, or from bilateral calculus. Or it may be unilateral, from stone in the pelvis or ureter, from kinking of the ureter owing to movable kidney, or from dragging or compression of the ureter by inflammatory adhesions or new growths. In a few cases no cause can be found.

Pathology.—In the earlier stages the kidney appears normal until it is slit up, when it will be found hollowed out more than usual, the papillæ being flattened, the cortex thinned, and the secreting tissue diminished in amount. As the distension becomes more advanced it affects either the kidney proper or the renal pelvis. In this way two types are produced. In the *pelvic* type the kidney does not become enlarged at all, but is perched, like a cap, upon a sac formed by the dilated renal pelvis. As this sac enlarges, the kidney diminishes in size, and in extreme cases its atrophied remains are scarcely noticeable. In the *renal* type the pelvis is not dilated, but the kidney itself becomes enormously expanded. It preserves its reniform shape, but becomes lobulated. On section it is found to be composed of large thin-walled cavities (dilated calyces) communicating with the renal pelvis, but separated from each other by thin fibrous

partitions (the remains of the columns of Bertin).

Symptoms may be absent. Patients with enlarged prostate, stricture, etc., may have their kidneys transformed into mere shells before any renal symptoms are observed. In these cases the kidneys are not enlarged. The most characteristic symptoms of hydronephrosis are pain and tumour. The pain may be a dull ache felt in the angle between the last rib and the erector spinæ muscle, or it may come on in sharp attacks resembling renal colic. Often the two types alternate. The acute attacks are not, as a rule, distinguished from a true renal colic, but generally they do not last so long, have no relationship to jolting or exercise, and are not accompanied by hæmaturia or strangury. The pain comes on slowly, and gradually increases in intensity. When it is at its height the patient experiences a sensation of great distension in his side, and often says that he feels "as if something must burst in him." The pain does not, as a rule, radiate to the groin or testicle, and the attack subsides gradually as it commenced. The tumour varies from a scarcely palpable kidney to an enormous cyst filling the greater part of the abdomen. A comparatively large hydronephrosis may be difficult to feel if it is flaccid. The tumour is reniform, smooth, painless, and not tender on palpation; it moves freely with respiration. As it increases in size it descends more and more below the lower ribs, fills out the loins, and may form a visible swelling in the lumbar region. During the attacks of pain it may be masked by muscular rigidity, and the kidney is then distinctly tender on pressure. The tumour may be constant or intermittent; in the latter case it is present during the attacks of pain, but when they cease the swelling disappears, and there is a greatly increased flow of urine. This is called an intermittent hydronephrosis. Polyuria is sometimes a noticeable symptom; it is more marked in bilateral than in unilateral cases.

Diagnosis.—It is most important to diagnose the condition early if the kidney is to be saved. This has been rendered possible by the use of ureteric catheterization and the X-rays. If a ureteric catheter is introduced into a dilated renal pelvis, the urine escapes in a continuous stream until the sac has been emptied. In this way an estimate of its capacity may be made. Another method is to inject sterile water through the catheter until

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the patient just begins to feel pain. The pain indicates that the kidney has been distended, and the amount of fluid necessary to produce this effect is noted. More exact information can be obtained by *pyelography*. When the renal pelvis is filled with a solution of collargol, thorium citrate, sodium bromide, or other substance opaque to the X-rays, and radiographed *in situ*, the shadow reproduces the form and size of the dilated pelvis. In the renal type the first changes are found in the calyces. They become club-shaped; normally they are trumpet-shaped. Later they tend to become pear-shaped, and the space between them is reduced to a minimum. In the pelvic type a large irregular shadow of the renal pelvis is seen; this has slight elevations on its outer side, corresponding to the stunted calyces.

Treatment and prognosis.—Treatment consists in removal of the cause. The possibility of bilateral hydronephrosis must never be lost sight of when dealing with stricture or enlarged prostate. In other cases, if a stone is present it should be removed, if the kidney is movable it should be fixed, or if the obstruction is due to a valve or kink it should be relieved by a plastic operation. It is only when the kidney is totally disorganized that a primary nephrectomy should be performed. In most cases the kidney recovers the greater part of its functional activity after the obstruction has been relieved. In aseptic cases the operative mortality after a plastic operation, or a primary nephrectomy, ought to be nil. If the plastic operation fails, a secondary nephrectomy may be necessary. Of course, in bilateral cases the risk is greater, and conservative measures are essential.

J. SWIFT JOLY.

HYDROPERICARDIUM.—Fluid may be poured into the pericardial sac in connexion with general œdema and may seriously hamper the heart's action. Its signs are those of pericardial effusion due to pericarditis, but it is generally overlooked. Cases have been recorded in which, after scarlet fever, a hydro-pericardium has been almost the only evidence of serous transudation. Rarely, as in elephantiasis, the exudate may be chylous.

FREDERICK LANGMEAD.

HYDROPHOBIA.—Hydrophobia, or rabies, is a disease of the lower animals, especially of canines—dogs and foxes—and of cats, which is communicable to man. The virus in infected

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animals exists in the saliva and other secretions as well as in the nervous system, and man becomes inoculated as the result of being bitten by a rabid animal. A comparatively small percentage (about 15 per cent.) of persons bitten develop the disease. Those who are bitten through wearing apparel have a very fair chance of escape. After a long period of quiescence, outbreaks of the disease have occurred in this country, and dog-muzzling orders for the districts concerned have had to be issued from time to time.

Symptomatology.—The period of incubation is about eight weeks on the average, but may be as long as two years; it is sometimes as short as two weeks, especially in children. The first premonitions of the disease are found in numbness and irritability of the wound. There is slight fever. The patient becomes depressed and filled with dread, and may have some difficulty in swallowing. He is hypersensitive to noises and light. In a day or two these symptoms become intensified; excitability and hyperæsthesia become extreme. Violent painful spasms occur, especially in the pharynx on every attempt to drink (hence the name of the disease), or as the result of any external stimulus. Severe attacks of dyspnoea, and of mental agony or actual mania, add to the distress of the disease. The fever generally rises to 102° F. This stage lasts three or four days and then passes into one of increasing paralysis, unconsciousness, heart failure, and inevitable death. Altogether the disease lasts about one week.

Treatment.—The wound inflicted by a suspected animal should be allowed to bleed freely and then cauterized with pure carbolic acid or other caustic. Once the disease is established, the treatment is wholly symptomatic. It consists in keeping the patient in a dark, quiet room as unmolested as possible. Inhalations of chloroform and injections of morphia will help to allay the spasms and agony.

The preventive inoculation introduced by Pasteur over thirty years ago should be tried if there is reason to fear that infection has occurred. It consists in inoculating on successive days a sublethal but gradually increasing dose of the virus which has been modified by transmission through a series of animals.

A curious hysterical condition may simulate rabies. This so-called *pseudo-hydrophobia* de-

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velops in persons some months after they have been bitten by an animal supposed to be rabid. There are depression and difficulty in swallowing, but there is no fever, and the disease lasts longer than rabies. It does not progress and is curable.

F. C. PURSER.

HYDRO-PNEUMOTHORAX (see PNEUMOTHORAX).

HYDROSALPINX.—A Fallopian tube distended by serous fluid.

Etiology and pathology.—Hydrosalpinx results from inflammation of the tube, which closes its end, and so prevents the escape of fluid secreted by the mucous membrane. The intensity of the inflammation determines the nature of the fluid; when this is serous, the infecting organism has died out. Distension occurs at the outer end of the tube, which becomes retort-shaped and seldom larger than an orange. Its wall is thin and translucent, and may be either adherent to surrounding structures or free.

Symptoms.—These are usually not urgent. Commonly there is a history suggesting old pelvic inflammation. The patient may complain of backache, dysmenorrhœa, and dyspareunia, and of pressure in the pelvis if the hydrosalpinx is large.

Sterility is inevitable if both tubes are closed, and may be the only cause of complaint.

Acute symptoms may arise from torsion of the mesosalpinx, an attack occurring indistinguishable from that due to the torsion of an ovarian cyst, or by infection of the hydrosalpinx from the bowel, with consequent acute inflammation.

Diagnosis.—The diagnosis of hydrosalpinx is difficult; the condition may easily be mistaken for an *ovarian cyst* or a *chronically thickened tube*. In the case of an ovarian cyst there is no history of pelvic inflammation, and the swelling is more spherical, and usually more movable; the thickened tube feels harder and less cystic.

Treatment.—If the symptoms are slight and the swelling small, nothing need be done. Otherwise the treatment is surgical; the tube should be removed, leaving the ovary undisturbed. When advice is sought on account of sterility, salpingostomy should be attempted.

J. P. HEDLEY.

HYDROTHERAPY.—The object of this article is to explain the principles involved in the external use of water for the purpose

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of treating disease, and thus to enable the practitioner to select the cases which may with advantage be sent to spas or bathing establishments. A classification of the various mineral waters, with details of their properties, is given in the article on SPA TREATMENT.

In view of the scepticism which is frequently expressed as to the value of hydrotherapeutic measures, it is necessary to consider briefly the principles underlying them. Water applied externally acts upon the nervous and vascular structures of the skin by means of two different forms of stimulus—mechanical and thermic. Thermic stimuli result from the temperature of the water used; mechanical stimuli are set up by the impact of sprays and douches, by the friction or massage which is an essential feature of many forms of bath, and, in the case of gas-containing waters, by the irritant action of the small bubbles which form upon the skin. A further stimulus may result from the chemical character of the water used, and is most evident in the case of the stronger saline waters. The actions of heat, cold, and friction upon the skin are matters of everyday occurrence, and their influence in maintaining the general health is of the utmost importance. A large proportion of the blood is contained in the capillaries of the skin—a proportion that is capable of great variation under the influence of stimuli which act through the nervous system upon the muscular coats of the arterioles. All stimuli, whether thermic or mechanical, act primarily upon the cutaneous nerves, which transmit their influence to the central nervous system; and by this means the body-heat is regulated. The tone of the circulation is also dependent to a great extent upon the reactive capacity of the cutaneous vessels. In addition, the circulation through the viscera and the nervous system may by the same means be profoundly modified, muscular power may be improved or diminished, the excretory functions of the skin stimulated, and metabolism influenced. Some authorities claim, not without strong evidence, that the capillary network possesses the power of rhythmic contraction, thus materially assisting the heart. Much experimental evidence might be quoted to prove all these points, but the experiments of Vinaj and Maggiori may suffice. These observers have shown that a cold bath increases the capacity for muscular work by 50 per cent., and that that capacity is more than doubled by a warm bath, gradually cooled, of several

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minutes' duration. Other observers have shown that by a course of baths nitrogen metabolism and excretion are increased, though a single bath has little influence in this direction. No other method of obtaining these effects has so wide a range of action or is susceptible of such complete control and adaptation to the needs of the patient. Minute attention to details is essential, however, if the best results are to be obtained, and it is to neglect of these details that failure, or partial failure, is generally to be attributed. The more elaborate procedures require a well-equipped bathing establishment, but excellent results can be obtained from the simpler ones in the patient's own home, though not, of course, in the case of mineral-water treatments.

The **simple immersion bath** is the most commonly used hydrotherapeutic measure, and its action throws light upon the more complicated procedures. It may be "full"—the patient immersed up to the neck; "three-quarter"—to the middle of the chest; or "half"—to the waist; the depth, as a general rule, depending upon the strength and reactive capacity of the patient. When there is great enfeeblement, or when a special local effect is required, such modifications as the sitz-bath and baths for the limbs only may be ordered. The temperature used varies within a wide range and modifies greatly the effect of the bath.

The effects of temperature as well as of other stimuli have been very carefully studied by Sonntag and others at the Shepherd's Bush Military Hospital, where hydrotherapeutic methods were widely and successfully employed during the War. For descriptive purposes the following is a useful range of temperatures:

Hot, 97°–104° F.; *neutral*, 92°–97° F.; *tepid*, 80°–92° F.; *cool*, 65°–80° F.; *cold*, below 65° F. Hot baths are stimulating in action but depressing in reaction; cold baths, on the other hand, are depressing in action but stimulating in reaction; while baths at neutral ranges of temperature have no thermic action on the cutaneous nerves or circulation and may consequently be prolonged indefinitely.

Full **hot baths** between 98° and 104° F. slow the pulse at first, but after a variable period it accelerates again, and the rate increases till a point is reached at which it remains constant for a time, when a series of fluctuations occur. Above 106° F. there is a paralytic dilatation of the cutaneous vessels from the start. In giving hot baths for therapeutic purposes the

face should be sponged with cold water first if the temperature is above 100° F., and a wet towel wrapped round the head; thus by reflex action the cerebral circulation is protected against violent circulatory changes. For local baths higher temperatures may be used; for instance, 108° F. for twenty minutes, reducing the duration as the temperature is raised up to 118° F., at which eight minutes is long enough. To counteract the late depressing effect of hot baths they should be followed by a cool or cold affusion, douche, or needle spray. Hot baths are useful for fatigue or exhaustion, but where this is at all extreme the cold application should be omitted and the patient should rest in bed afterwards. For antispasmodic effects the bath should be given about 102° F. and prolonged to fifteen or twenty minutes; it will be found useful in spastic conditions, arterio-sclerosis, nephritis, and plumbism.

Neutral baths produce no thermic effect, but shut off all thermic and mechanical stimuli from the skin and allow the nervous and circulatory organs, which are always in a state of activity corresponding to the environmental impressions, to rest. They are useful in shell shock, traumatic neurasthenia, extensive burns, delirium and mental excitement, exhaustion, and fever. The effect is enhanced in the "pool bath" by keeping the room darkened. This (92°–97° F.) is the range of temperature usually employed in the immersion baths of certain spas, but a douche of water at a higher temperature is given under the water during the bath. Both hot and neutral baths are given with advantage in muscular rheumatism and fibrositis, the temperature depending usually on the character of the water employed; a lower temperature is used if chemical stimulation may be expected from the character of the water or if mechanical stimulation is also to be combined.

The full **tepid or cool bath** (92°–65° F.) is seen in its most potent form in those baths through which a continuous stream of water runs; this is a characteristic feature of spas possessing thermal radioactive waters. At a temperature below 90° F. the bath is stimulating if not too prolonged and if the patient's reactive capacity is good. The effects are modified by the nature of the water. If the bath is fed direct from the spring and contains natural gases, the bather breathes these gases at the surface of the water, and potent influences may thus be produced. The general effects of these cool baths may be deduced from those of the

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Brand bath, in which friction gives place to the stimulation due to the brisk exercise of swimming or splashing about, and is supplemented by the effect of the gases and mineral constituents upon the nerve-endings of the skin. This is not to say that such constituents are absorbed by the unbroken skin—of which there is no proof, however probable it may be that the gases at least may enter the circulation by osmosis—but they will get into the blood-stream much more rapidly by being inhaled from the surface of the water, and any other mode of ingress may be ignored. That the radioactive gases do produce some part of the effect of the bath by being thus inhaled is certain.

The cool bath in its therapeutic aspect, as distinct from the popular "cold tub," the stimulating effect of which need only be mentioned, is best exemplified by *Brand's method* of treating enteric fever. It is applied as follows: The bath is filled to three-fourths of its depth with water at a temperature varying from 90° to 65° F., the higher temperature being used in the first bath, and progressively lowered about 5° each time until the lower limit is reached. Where possible the bath is brought to the bedside; it is hardly necessary to say that an enteric patient cannot walk to the bathroom. The patient is lifted in very gently by two attendants and his head supported by a pillow. Friction of the whole body is then commenced and kept up throughout the time of immersion—a most important feature which must never be omitted. The duration of the bath is from five to twenty minutes; a short immersion in cool water is more stimulating though less heat-reducing than a longer bath at a higher temperature. The effect of the friction is to cause redness of the skin and thus to promote heat-loss, chilling or collapse being at the same time prevented. Shivering or blueness of the extremities is not a contra-indication to the use of the bath, but an incentive to more energetic friction. The bath may be given three, four, or more times in the twenty-four hours, according to circumstances. The effect of this treatment is often most striking, and statistics show a marked reduction in mortality in those institutions where it has been adopted. Its effect is mainly to stimulate and strengthen the vital organs, enabling them to throw off the toxins of the disease; it also acts as an antipyretic which is free from risk, since it is at the same time a powerful cardiac stimulant.

The foregoing description applies to the effects of baths of the waters of any of the better-known British spas, and to sea-water baths. The more saline and consequently stimulating waters have their effects modified by temperature, dilution, etc.; while the less mineralized waters, which in this country are usually more gaseous, have their sedative action qualified by exercise in the water, as for instance in swimming-baths, and by douching and massage, and thus become stimulating. The capacity possessed by hydropathic procedures of thus being modified to suit individual patients is of the greatest value.

Certain special baths deserve description. The so-called **Nauheim system** depends on the use of a saline water which, given in the earlier baths in a dilute form and without gas, is gradually increased in strength and gaseous content with successive baths, and at the same time is lowered in temperature. Thus a mild sedative bath, having a soothing effect on the circulation and at the same time a mildly constrictor effect, gradually gives place to one which powerfully stimulates the vasomotor mechanism and the system generally. In skilled hands the artificially prepared bath, available at most British spas and many sea-side places, appears to give results quite equal to those actually obtained at Nauheim, though falling short, perhaps, of the results claimed for that particular spa.

Moor baths, prepared by the admixture of a specially prepared peat with the mineral water to a prescribed consistency, have a sedative effect, and are particularly comforting in cases of muscular rheumatism, fibrositis, and some forms of arthritis. They are also useful in certain kinds of eczema and psoriasis and as a nerve sedative. They are given at temperatures of 90° to 105° F., and are followed by a cooler bath or spray.

Douches form an important feature of all bathing treatments; their effect is generally stimulating, and they may be given alone or as part of a general bath. The simplest douche consists of a jet of water played upon the body from a distance at a particular temperature and pressure and for a specified time. The effect is to set up a powerful vibration in the structures treated, penetrating more deeply than massage, and it is thus of benefit in chronic rheumatic affections, in promoting absorption, and, especially when played upon the spine, in stimulating the nervous system. A common modification is the **Scotch douche**, which is

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given in the same way but at varying temperatures, a few seconds at a high temperature being followed by a sudden change to a lower, and back again, for the number of times prescribed; the temperatures and the exact duration of each alternation need to be specially adapted to each case. The **contrast bath** may be mentioned here. It consists in immersing first in hot and then in cold water the part of the body to be treated, repeating this for ten or fifteen minutes. A useful range of temperature for the two foregoing methods is as follows:—Scotch douche, 110°–115° for 30 sec., 50°–65° for 15 sec. (Sonntag); contrast bath, 116° for 30 sec., 40°–50° for 15 sec.

The **needle douche** is too well known to call for particularized description; generally it is warm at first and is gradually cooled, but it may be varied in any way. It is a pleasant stimulant to the peripheral nerves, and, if the cold phase is not emphasized too much, may cause drowsiness and sleep as a reaction.

Massage douches are popular methods of treatment. They are given by one or two attendants who massage the whole body while a stream of water is playing upon it from a hose-pipe hanging over the operator's shoulder. The Aix douche, which was the original, is given with the patient seated upon a stool or lying on a bench. At Vichy was introduced the modification of allowing him to lie upon a water-bed while sprays or jets of water fall upon him from above at the will of the operator. At Buxton a further modification was introduced: the patient lies in a shallow pan of water while being massaged and douched, the advantage being that the muscles are relaxed through the body being supported in the water, instead of being contracted in maintaining the upright or reclining position on a chair or bench. The advantages of douche massage by either method are obvious; for stiffness of muscles or joints there is probably no mode of treatment which will compare with it, while it is very effective in stimulating the peripheral circulation and metabolism.

Certain special douches call for more detailed consideration. The **under-water douche** is given beneath the surface of the water in which the patient is immersed, either to the joints, to the spine and lumbar region, or to the abdomen. In either case its effect is that of a deep massage with a vibratory character, of which the surrounding water appears to mitigate the force to some extent without lessening the general effect.

The **whirlpool bath**, which acquired considerable popularity during the War on account of its effect in relaxing stiffened joints and tissues, is an immersion bath for the whole or part of the body, in which a continuous movement is kept up either by the inflow of a stream of fresh water or by a turbine which, continually revolving, produces great agitation of the whole bath and an "effleurage" effect on the part immersed. In another form compressed air is driven in and a continual effervescence thus obtained. In general effects both forms are similar to the douche massage methods just described.

The **Plombières douche** has come rapidly into fashion during recent years, thanks to the excellent results it gives when used with due discrimination, not only in membranous colitis but in intestinal stasis and disorders arising therefrom. It is administered by means of a soft rubber tube 8 or 10 in. long inserted well into the rectum; through this tube water of which the quantity, temperature, and pressure are to be definitely prescribed is injected. Under favourable conditions it will reach the higher parts of the colon, and even the cæcum, as may be ascertained by abdominal examination. It is retained for a few minutes and then evacuated in the usual manner. Its action is to remove faecal accumulations, mucus, etc., which have often been retained in the recesses of the colon for a very long time despite the use of aperients, and to cleanse and soothe the mucous membrane, thus restoring its normal functions. These effects are modified to some extent by the kind of mineral water used. Thus, thermal radioactive waters like those of Plombières, Buxton, and Bath are non-irritating and have a soft, silky feeling to the skin, which renders them peculiarly soothing to an inflamed mucous membrane. The precise part which their radioactivity may play has yet to be demonstrated, but it is probably important. Saline waters like those of Châtelguyon, Llandrindod, etc., are slightly more stimulating, and for the former is claimed special virtue on account of the magnesium chloride it contains. With these waters must be included the sulphur waters of Harrogate, etc., whose action in this respect depends in great measure upon their saline content; how far the sulphur exerts an antiseptic action or a special effect on the mucous membrane has, like the effect of radioactivity, still to be demonstrated.

Another form of douche for internal use is

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employed for the respiratory tract, and is more correctly a **spray of finely atomized mineral water**. Besides the local action on the mucous membranes of the nose and throat, the state of fine division to which the water is reduced enables it to be drawn deeply into the lungs, and this is of special value where radium emanation is present.

Local baths.—In the **sitz-bath** the patient is placed with the feet outside the bath, the water being only deep enough to cover the hips. The **hot sitz-bath** reduces visceral and pelvic congestion, and is useful in menstrual disorders, chronic pelvic inflammation, congested stricture, etc. The **cold sitz-bath** is tonic and sometimes anodyne, and is of use in atonic conditions. It should be brief in duration, and constant friction should be kept up.

The **hot foot-bath** has a derivative effect in congestive headaches and other conditions. The **cold foot-bath**, of brief duration and followed by friction, often proves effective in insomnia.

Packs.—These are among the simplest of hydrotherapeutic methods. The most important form is the **whole-body pack** for conditions of fever or nervous excitement. A couch is prepared by spreading over it one or more blankets, and on this is laid a sheet wrung out of water at about 75°–85° F. The patient lies down and is closely wrapped up, first in the sheet, which must be in close contact with his body, and then in the blankets, all being well tucked in. Hot-water bottles may be placed around the patient if necessary; and after the pack, which may last an hour, a needle-bath is often given. **Local packs**, such as those for the throat, chest, or individual joints, are applied according to the same principles.

Packs of **peat**, or **mud**, are also used, and have the effects of a hot poultice modified in some measure by the material used; they are usually followed by douching. **Mustard packs** are practically mustard poultices, and need no further description.

The **Turkish bath**, while not strictly hydrotherapeutic, is closely associated with such procedures. Its distinctive feature is the induction of perspiration by exposure to hot air at 110°–200° F. This is followed by soaping and shampooing of the whole body, after which a cool or cold douche is given, and a plunge if desired. Its effects are cleansing and refreshing, while it stimulates the circulation

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and aids elimination. The **Russian bath** is similar to the Turkish, but steam is used instead of air, and the temperature is not so high—110° to 120° F. Needless to say, neither should be used in conditions of cardiac debility. The **vapour or Borthollet bath** consists of a cupboard filled with steam at a temperature of 105°–120° F.; in this cupboard the patient sits or has a portion of his body enclosed. The effect is diaphoretic, and the bath is generally followed by a douche or immersion bath. The **electric-light bath** is similar in effect, but the heat is the dry heat obtained from numerous electric lamps (see LIGHT TREATMENT). In the **Greville electric-heat bath** the heat is obtained from rows of thin iron wires heated to a dull redness by the passage of an electric current, the dark heat rays being used in contrast to the preceding method.

C. W. BUCKLEY.

HYDROTHORAX.—A collection of transuded fluid in the pleural cavity. It differs from pleurisy with effusion in that it is not due to inflammation, but the signs are identical. Usually it forms part of a general anasarca, and it is therefore accompanied by ascites and oedema and by the signs of one or more of the causes of general oedema (see OEDEMA). In cardiac cases it is more often right-sided. More rarely it is due to pressure on the root of the lung on the affected side by malignant growth or by glands. In these circumstances, or as the result of blockage of a main lymphatic channel by *Filaria bancrofti*, or of other cause, the fluid may be chylous. The effusion is seldom so great as in inflammatory cases and, apart from the chylous form, is clear; it contains no flocculi of fibrin, but few cells, which are chiefly endothelial, and the specific gravity is generally below 1015. The treatment is that of pleurisy with effusion (see PLEURISY).

FREDERICK LANGMEAD.

HYDRURIA (see URINE, VARIATIONS IN AMOUNT OF).

HYMEN, DISEASES OF (see VULVA, DISEASES OF; AMENORRHEA).

HYOSCINE, POISONING BY (see POISONS AND POISONING).

HYOSCYAMINE, POISONING BY (see POISONS AND POISONING).

HYOSCYAMUS, POISONING BY (see POISONS AND POISONING).

HYPERKERATOSIS

HYPERACIDITY (see STOMACH, FUNCTIONAL DISORDERS OF).

HYPERÆSTHESIA, VISCERAL (see STOMACH, FUNCTIONAL DISORDERS OF).

HYPERCHLORHYDRIA (see STOMACH, FUNCTIONAL DISORDERS OF).

HYPERIDROSIS (see SWEAT-GLANDS, AFFECTIONS OF).

HYPERKERATOSIS.—This term more correctly describes a symptom than a disease. It is used for those conditions where the horny layer of the skin is unusually developed, and is met with in its most extreme degree on the palms and soles. In addition to the thickening, there is a tendency for fissuring to develop. When the observer is confronted with such appearances he should first attempt to find evidence of disease elsewhere, as this may afford a clue to the diagnosis.

One of the commonest varieties is associated with **eczema**. Here the keratosis is usually symmetrical, ill defined, tending to fade imperceptibly into the normal surrounding skin; it is peculiarly chronic, and, as in eczematous processes generally, there is distinct tendency to relapse. Both palms and soles may be involved at the same time. If closely examined, evidence of vesiculation can usually be detected. *Treatment* is usually unsatisfactory and begins with the removal of scales mechanically by pumice-stone; then by applications containing salicylic acid and oil of cade. The best results are obtained by X-rays, the affected area being subjected to a dose varying from half to two-thirds of a pastille, repeated if necessary after an interval of three weeks.

Hyperkeratosis is also found associated with such conditions as psoriasis, ringworm, and syphilis. In **psoriasis** the diagnosis can frequently be made by the discovery of typical lesions on the elbows or knees. There is a special tendency to exfoliate, and in any case, if the surface scales be removed, the underlying skin is found red and smooth as in psoriasis elsewhere. *Treatment* is similar to that employed for eczema, X-rays being of peculiar service.

When the condition is due to **ringworm**, **greasy** difficulty may be found in arriving at a correct conclusion. Scrapings should be made and examined microscopically for fungus in the usual way. Sometimes tiny pustules may be detected, especially at the edges of the patch. *Treatment* is carried out by chrys-

arobin ointment or by an ointment consisting of benzoic and salicylic acid 15 gr. each, vaselin 1 oz.

In both **secondary** and **tertiary syphilis** the palms may be affected. In the former, red, scaly, lenticular papules are present and represent a secondary eruption. In the later stages either one or both palms may be involved. The appearances are distinctive, the edges of the eruption being demarcated in a peculiarly sharp fashion. Diagnosis may be established on this feature alone, or confirmation may be obtained by a positive Wassermann reaction or by other evidence, such as glandular enlargement. Intensive *treatment* by salvarsan and mercury should be carried out.

Arsenical hyperkeratosis.—Hyperkeratosis is frequently found after arsenic has been taken internally for a prolonged period, a possibility which should always be borne in mind in cases in which this drug has been administered. Pigmentation, especially of the abdomen, frequently coexists.

A form of keratosis is met with in elderly people about the face and on the backs of the hands as multiple, greasy, discoloured, warty formations—**senile keratosis**. Apart from the unsightly appearance, its chief importance lies in the possibility of malignant degeneration. If considered advisable, the lesions may be removed by radium or X-rays.

For keratoderma blenorrhagica, see GONORRHEA.

II. MACCORMAC.

HYPERMETROPIA (see REFRACTION AND ACCOMMODATION, ERRORS OF).

HYPERMNESIA (see MEMORY, DISTURBANCES OF).

HYPERNEPHROMA (see KIDNEY, TUMOURS OF; and SUPRARENAL GLANDS).

HYPERPITUITARISM (see PITUITARY GLAND, AFFECTIONS OF).

HYPERSECRETION, GASTRIC (see STOMACH, FUNCTIONAL DISORDERS OF).

HYPERTONUS, GASTRIC (see STOMACH, FUNCTIONAL DISORDERS OF).

HYPERTROPHIC OSTEO-ARTHROPATHY (see OSTEO-ARTHROPATHY, PULMONARY).

HYPERTROPHIC STENOSIS OF THE PYLORUS (see PYLORUS, CONGENITAL HYPERTROPHIC STENOSIS OF).

HYPOCHONDRIASIS

HYPNOTICS, POISONING BY (see POISONS AND POISONING).

HYPNOTISM (see PSYCHOTHERAPY).

HYPOCHLORHYDRIA (see STOMACH, FUNCTIONAL DISORDERS OF).

HYPOCHONDRIASIS.—Many a healthy person has a complex around which his thoughts revolve. For example, in adolescents the complex is most frequently sports and games; in others, motoring, photography, music, etc., form complexes. There are others, again, in whom the complex is the state of their health. Such a one is ever looking in the mirror to examine his tongue or his throat. He feels his pulse and takes careful notes of the state of his bowels and his urine. He studies medical literature, is morbidly interested in new forms of diet, of treatment, of general hygiene. Slight disorders of the bodily mechanism fill him with uneasiness, and he is continually resorting to drugs, digestive powders, tablets, tonics, and patent foods. He is, in fact, a hypochondriac.

A more pronounced type is the man whose sole interest in life is his health. He is unable to do his daily work, and loses all social interest. He imagines that vague pains in his abdomen are the beginnings of cancer, certain spots on his skin are the signs of syphilis; and every day, as fresh signs arise, fresh diseases are diagnosed. Finally, there are cases where the patient has the delusion that he is affected with some definite disease. He is firmly convinced that he has cancer of the throat, or that there is a tumour in his abdomen, or that his bowels never act. In the last example it is easy to produce evidence to the contrary, but that has no effect upon the delusion. Such patients are insane, and as these delusions have a depressing effect they are classified as hypochondriacal melancholia.

Men are more affected than women, and the age of onset is, as a rule, late adult life. Hypochondriacal melancholia must not be confounded with hysteria, nor must it be mistaken for neurasthenia, which is a condition of exhaustion having, as a rule, definite sensory symptoms such as headache, spinal pain, and sensations affecting the visceral organs. It is possible for the neurasthenic to have hypochondriacal ideas, but they do not form the definite delusions of the hypochondriacal melancholic.

Diagnosis.—The principal point in diag-

HYPOSPADIAS AND EPISPADIAS

nosis is to be certain that the illness complained of is a delusion and not a fact. If the usual clinical methods are unavailing to clear up the case with certainty—and this is a rare event—time will usually do so. It is possible for hypochondriasis to exist as a complication of organic diseases. For example, it is often met with in association with general paralysis of the insane. A careful clinical examination is the only safeguard against error.

Treatment.—In the milder cases every effort must be made to direct the train of thoughts into other channels. Hobbies, games, travel, or some special line of work may each be tried, and if an earnest attempt is made by the physician to understand his patient and to discover something in which he will really be interested, success may be attained. At the same time, minor ailments which serve to bolster up the condition may be detected, and with their cure the hypochondriasis may cease.

As in hypochondriacal melancholia the state is one of fixed delusion, little can be done to effect a cure. It must be remembered that these patients are stated to be very suicidal (though this has not been my experience), and precautions must be taken accordingly. As a rule, patients of this class are best treated in a mental hospital, for they are difficult to manage in their own homes and most trying to their relatives. The condition does not tend to shorten life.

R. H. STEEN.

HYPOPHYSIS (see PITUITARY GLAND, AFFECTIONS OF).

HYPOPITUITARISM (see PITUITARY GLAND, AFFECTIONS OF).

HYPOPYON ULCER (see CORNEA, AFFECTIONS OF).

HYPOSECRETION, GASTRIC (see STOMACH, FUNCTIONAL DISORDERS OF).

HYPOSPADIAS AND EPISPADIAS.—*Hypospadias* results from imperfect fusion of the lips of the uro-genital sinus so that the floor of the urethra is missing to a greater or less extent. Three degrees are recognized. In the first the urethra opens at the base of the glans penis, there is no frænum, and the prepuce, instead of surrounding the glans, hangs around it like a hood, being absent below. Though inconvenient, this degree is of little importance and calls for no treatment. In the second or *penile* form the urethra as a closed

HYSTERIA AND HYSTERO-EPILEPSY

is taken. Anæsthesia, when established, may be complete or partial; it may involve all forms, both cutaneous and deep, or it may be dissociated. As with all hysterical symptoms, it corresponds to the patient's idea or conception of what such anæsthesia entails; thus it never conforms to anatomical distributions of peripheral nerves. Its segmentation, moreover, is not that of spinal disease.

(d) **The reflexes.**—The condition of the cutaneous reflexes in hysteria is variable. The plantar reflexes are usually typically flexor, but sometimes they are difficult to elicit at all. The tendon reflexes also vary much, being often exaggerated; they have been recorded as temporarily absent, too, but it is difficult to accept their absence as being due to hysteria alone. A form of spurious ankle-clonus has been so commonly met with in hysteria that it has been supposed to be characteristic of that neurosis; this is not, however, the case. The organic reflexes are, as a rule, intact, but occasionally they are defective. The pharyngeal reflex is often diminished, and loss of the corneal reflex is stated to occur.

(e) **Sympathetic, vaso-motor, trophic and secretory symptoms.**—The reality of hysterical fever has been as much denied as accepted. Trophic changes in the skeletal musculature in longstanding cases of hysterical palsy have often been seen. Certain skin affections of a trophic nature—e.g. multiple neurotic gangrene—seem sometimes to develop on a basis of hysteria. Vaso-motor phenomena are common. Hysterical anorexia is well recognized. A large number of miscellaneous sympathetic and vaso-motor symptoms have been assigned to hysteria, apparently merely because no other adequate cause for their appearance has been discovered.

(f) **Hystero-epilepsy, somnambulism, fugues, dissociated personality.**—In a high percentage of cases hysterical attacks in one or other form are met with. They may range from a mere faint to elaborate and prolonged automatism with wild convulsions, passionate attitudes, and complex exhibitions of subconscious activity. The attack is to be considered as a manifestation of a psychological disturbance; in Janet's words, "a simple idea, a system of images which has separated from the totality of consciousness and has an independent development, brings about two things—a blank in the general consciousness, which is represented by amnesia; and an exaggerated and independent development of the emancipated idea." The movements, then, are purposive

and readily separable from the crude convulsions of ordinary epilepsy, which, for that matter, can be distinguished also by other features, such as tongue-biting and involuntary micturition.

The slightest, mildest forms of hysterical attack may not be easily differentiated from petit mal. Moreover, the automatism that may follow an attack of grand mal or petit mal may be indistinguishable from the hysterical form, unless all the facts of the case are carefully investigated and observed.

There is no dividing line between the hysterical attack and the more elaborate fugue. The latter is a sort of ambulatory automatism, of varying duration. It, in its turn, is not far removed from the more complicated states of mental dissociation of the neurosis, in which separate "existences" and "personalities" are formed.

Prognosis.—The prognosis in hysteria is complicated by various factors. As far as individual symptoms are concerned, there is little doubt that it is good—i.e. under suitable treatment they will vanish, and sometimes in a dramatic fashion. This is true more particularly of the obvious somatic manifestations of the neurosis—palsies, contractures, involuntary movements, anæsthesias, etc. Similarly, hysterical attacks can be cut short by a variety of methods, and in this sense the immediate prognosis is, as already remarked, good. When we come to the more complicated manifestations, however, the physician should remember that the outlook is not so satisfactory; for they lead to the question of the so-called hysterical constitution, and to discussion of the problem whether there is not an underlying predisposition, a prepared soil from which the neurosis springs. If this be so, the question of prognosis becomes more serious, inasmuch as the possibility of modifying or transforming or eradicating this predisposition is by no means certain. It has already been noted that in hysteria transference of symptoms is apt to occur, and often enough they may be made to disappear only to recrudescence at another time or in another guise. The view that we take of the prognosis depends in a sense on our views of the etiology, and it is an interesting fact that not a few workers on the subject have vaunted their own particular theory of the disease because of the good results they obtain when treatment is carried out along lines which they consider are suggested by their theory.

Speaking generally, however, it may be said

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that many cases make good and permanent recoveries, while others become inveterate in spite of every conceivable form of treatment.

Treatment.—This may be divided into two sections—(1) physiotherapy, (2) psychotherapy.

1. **Physiotherapy.**—First may be mentioned isolation and the Weir-Mitchell treatment. The patient is removed from his immediate environment and no visitors are allowed, with the exception of the doctor and the nurse. It may be desirable, also, to stop letter-receiving and letter-writing for the time being. The patient rests in bed, and is given general massage at regular intervals, once or perhaps twice daily. He is fed with milk and otherwise up to the limit of his digestive powers. By way of furthering this general physical treatment, it is often a good plan to give baths daily, or three times a week—e.g. sulphur or Droitwich baths. General electrical treatment, also, at regular intervals, may benefit the general condition; under this head faradic baths, high-frequency and static treatment, and central galvanization may be specified.

In regard to individual symptoms, it is often desirable to apply treatment locally. For flaccid palsies of whatever kind, local faradism and local massage will prove beneficial in a large number of cases. Sometimes a single application of the faradic current, or of the high-frequency effluve, or of static sparks, will be sufficient to bring the power back to a palsied group of muscles. Similarly, electrical treatment may be utilized with advantage in cases of involuntary movement—tremors, "chorea," blepharospasm, etc. Anaesthesias often disappear at once under such methods. Hysterical contracture is sometimes more difficult to treat by these physical methods; occasionally a whiff of chloroform may be administered to relax it, and this may be done, also, for involuntary movements which fail to yield to other treatment. Hysterical attacks can usually be cut short by old-fashioned methods such as throwing cold water on the face; firm pressure on the supra-orbital nerves may suffice, or a strong faradic current applied to various regions. In severe and prolonged crises a hypodermic injection of apomorphine, to induce vomiting, is sometimes peculiarly efficacious.

Drugs are generally of little use in hysteria, except in the treatment of certain symptoms. If hysterical attacks are violent or frequent,

the exhibition of one or other of the bromides will be serviceable. Sometimes a more powerful sedative or hypnotic, such as hyoscine, may be indicated.

There is no doubt, however, that unless the patient's general nervous nutrition is such as to call for nerve foods and nerve tonics, the less the hysterical patient has to do with drugs the better. And there is no doubt, either, that, except in those cases in which the above-described general treatment acts by improving the patient's metabolism as a whole, no forms of physical treatment are specific in themselves, but act merely as stimuli of a psychical nature—in other words, by suggestion. In fact, the whole armamentarium of physical methods is of quite secondary importance compared with treatment carried out along psychotherapeutic lines.

2. **Psychotherapy.**—There are several varieties of psychotherapy that are applicable to hysteria.

(a) *Treatment by suggestion.*—Included in this category are suggestion in the hypnotic state and suggestion in the waking state. To deal with the latter first: The patient is put into a condition of repose, of complete muscular and mental relaxation, as far as that is possible. To aid the production of a state of mental abstraction the physician talks in low, little modulated accents, and then proceeds to utter appropriate suggestions bearing on the patient's case. In the case of hypnosis the physician begins by suggestions which have a bearing on the production of a sleepy state, and this can be obtained by a variety of methods. It is important for the profession to realize that the patient is not actually asleep, or anything like asleep; it has been repeatedly pointed out that the deepest hypnosis may be induced with the patient's eyes open throughout. When the patient is in the hypnotic state his suggestibility is increased, his memory field is widened, and, by suggestion, effects can be produced on organs that are normally not within full voluntary control. Whether the condition be one of hypnosis, or merely of mental relaxation in the waking state, it is undoubtedly the case that appropriate suggestions may instantly, or in due course, modify, transform, or inhibit the symptoms. Needless to say, a *sine qua non* for the success of the procedure is that the practitioner should have confidence in himself and in his ability to impress his personality on the patient, as well as in the form of treatment he is adopting.

ICHTHYOSIS

(b) *Treatment by so-called rational psychotherapy.*—Under this heading are included the various procedures which aim at re-educating the patient from a mental standpoint, at teaching him to look in an altered way at the phenomena which have produced his "disease." He, for instance, may associate his symptoms with certain definite psychical and emotional disturbances—a shock, a fright, a strain or stress, and so on. When an idea in association with an emotion gives rise to morbid symptoms, the physician, analysing the facts minutely, endeavours to change or modify the old associations by linking one or other of their components to fresh ideas, harmless ideas; and so he, as it were, disarms the old association, which has been potent for mischief. He reasons with his patient, appeals to the intellectual side of him, takes him fully into his confidence and explains the significance of the symptoms, and thus helps him to "work out his own salvation." Treatment of this sort presents a distinct advance on "suggestion," for there is at least an attempt to get at the foundation of the trouble, whereas in the latter method there is no special endeavour at all to reach the pathological factors producing the neurosis. Not mere reasoning, not a mere cold intellectual process, however, suffices; the practitioner will get the best results if he imparts to his logical and analy-

tical exposition the influence which resides in a simultaneous appeal to the emotional side of his patient's nature.

(c) *Treatment by psycho-analysis.*—This term is here used in its technical sense, signifying the method developed by Freud for the examination and treatment of all sorts of "functional" nervous and mental disorders. As has been already noted, Freud holds, in common with Janet, that, as the result of dissociation, certain systems of ideas or complexes are repressed, yet remain in the unconscious mind as morbid factors. The aim of psycho-analysis is to recover these hidden complexes. A good part of the difficulty in psycho-analytic treatment consists in the fact that these complexes are found to be linked to thoughts, ideas, and desires of which the patient may be consciously ignorant. Once they are reached, however, and, in the process of reaching them, the mechanism of their conversion into, or rather of their production of, the symptoms is explained to the patient, and it is often found that their *raison d'être* then disappears. Psycho-analysis aims at revealing the cause of the hysterical symptoms, and for that reason alone, if for no other, it must be regarded as essentially a scientific method. Whether the method justifies all that is claimed for it by its ardent advocates is debatable.

S. A. KINNIE WILSON.

ICHTHYOSIS.—A congenital malformation of the skin characterized by excessive thickening, dryness, and scalliness of the horny layers.

Etiology and pathology.—The affection is congenital and runs in families, often affecting children of one sex only, but the actual cause is unknown. Persistence of the embryonic epitrachial layer of the skin is a suggested explanation. An excess of cornification of the cells of the horny layer and interference with the action of the sweat and sebaceous glands are essential features.

Symptomatology.—There are various degrees of ichthyosis, and to some of them special names have been given. A certain amount of unnatural dryness and roughening of the skin, termed *zeroderma*, is very common and scarcely calls for treatment. *Keratosis pilaris*

is a mild follicular ichthyosis giving rise to a nutmeg-grater roughness on the extensor surfaces of the limbs. *Ichthyosis simplex* is characterized by a general dryness and scalliness chiefly of the extensor surfaces of the limbs, with accentuation of the natural lines of the parts and a dirty-brownish colour of the skin. The flexor surfaces are often unaffected, and sweat secretion, which is diminished or abolished in other parts of the body, may be exaggerated in the usual sweat areas. When the face and scalp share in the process the former has a dry, shrivelled appearance and cracks form at the angles of the mouth, the scalp is scaly, and the hair dry and lustreless. The palms and soles are rough and hard, or sometimes polished, with marked accentuation of the furrows. *Ichthyosis hystrix*, or

IDIOCY, AMAUROTIC FAMILY

IDIOCY, IMBECILITY, ETC.

porcupine skin, which some authors classify as a linear or systematized nævus, occurs in the form of patches or bands of warty pigmented growths. Congenital foetal ichthyosis, or "harlequin foetus," and localized ichthyosis of the palms and soles—a congenital and familial affection—are generally classed with ichthyosis, although their relation to it is doubtful. The subjects of ichthyosis may experience no more than a certain amount of inconvenience, but the majority suffer from constantly recurring attacks of eczema owing to the peculiar susceptibility of the skin to chill or other external irritant. Many of them also suffer from chronic bronchitis or asthma.

Diagnosis and prognosis.—The importance of diagnosing the ichthyotic state underlying a case of eczema is obvious, and the harsh, dry skin is sufficiently characteristic to prevent confusion with other skin diseases. As a rule, ichthyosis is not noticed until several months after birth, and does not become pronounced until the end of the first or second year of life. The condition may then increase until about puberty, when it tends to improve, but nevertheless persists through life.

Treatment.—Although ichthyosis is a permanent malformation, and as such cannot be removed, much can be done to alleviate the symptoms. Thorough lubrication of the skin with bland oily substances such as olive oil, lanolin, vaselin, or glycerin and water, after a daily hot bath, will tend to prevent the excessive dryness and scaling of the skin. Internally, thyroid gland often has a marked temporary effect. Cod-liver oil and tonics are beneficial. Eczema must be treated with the remedies appropriate to that disease. If the scaling is very pronounced, salicylic acid may be added to the ointment, and salicylic plasters are of service in ichthyosis hystrix. The patient should be warmly clad and should be careful to avoid chills.

S. E. DORE.

ICHTHYOSIS LINGUÆ (see TONGUE, SYPHILIS OF).

ICTERUS (see JAUNDICE).

ICTERUS GRAVIS (see LIVER, ACUTE YELLOW ATROPHY OF).

IDENTIFICATION (see POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES).

IDIOCY, AMAUROTIC FAMILY (Waren Tay-Sachs' Disease).—This disease, which is limited to the Hebrew race, usually affects

two or more children of the same family, but does not occur in successive generations. It is characterized by a cessation and then failure of mental development, progressive weakness of all the muscles of the body, and rapidly-oncoming blindness. It leads to a fatal termination within the second year of life. Its pathology is a curious form of degeneration of all the nerve-cells of the body, including those of the retina; it is to the latter that the blindness is due. The chief symptoms, which appear during the first six months of life, are—listlessness and apathy, which increase gradually until the child is in a state of complete amnesia; progressive paresis of the limbs, which may be either spastic or flaccid, and, later, weakness of the muscles of the trunk so that the child is unable to sit or hold up its head; rapidly-developing blindness, with the characteristic appearance of a cherry-red spot at the macula lutea and optic atrophy; and a marasmus that progresses till death. Convulsions may occur, and in several cases nystagmus, squint, and hyperacusis have been observed. No treatment has been of any avail in arresting or checking the disease.

A *juvenile familial form* has been described by Spielmeyer, in which the pathological changes are very similar. This is not limited to the Jewish race; it commences later in childhood, and, though blindness develops, there are not the same changes at the macula lutea. There is always progressive mental failure, and convulsions are common. The course is longer, but it usually terminates fatally within three or four years.

GORDON HOLMES.

IDIOCY, EPILEPTIC (see EPILEPTIC INSANITY).

IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, MORAL IMBECILITY.—The most generally useful definitions of idiocy, imbecility, feeble-mindedness, and moral imbecility are those employed in the Mental Deficiency Act, 1913 (see article on that subject). According to these, the idiot is one who gives no sign of intelligence, the imbecile one who shows signs of a rudimentary intelligence, and the feeble-minded person one who displays more intelligence than the imbecile but is yet considerably below the intellectual level of the normal individual. The moral imbecile is in a class by himself, and is essentially one who is wanting in appre-

IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, MORAL IMBECILITY

ciation of the difference between right and wrong—a want inherent in the patient, and not the result of defective education.

Etiology.—The causes of congenital mental defect are often obscure. Mental and nervous diseases, particularly epilepsy, in forbears and collaterals, are said to be more than usually frequent in the family histories of defective patients. Consanguinity of parents, their alcoholic habits, and infections—especially syphilis—occurring in them before or at the time of the conception of the defective child, have been put forward as causes. Accidents, emotional shocks, infections and other diseases happening to the mother during gestation have also been regarded as bearing a relationship to the defective mental growth of the child. At parturition damage may be done to the infant's brain, by forceps or by a prolonged labour, which may interfere with its subsequent development. When the child has acquired independent existence, convulsions, meningitis, encephalitis, and various other infections and intoxications may stop or delay growth. There is reported to have been a falling off of the number of cases of idiocy associated with endemic cretinism since the hygienic conditions of the places in which it occurs have been improved. In sporadic cases it has been thought that a thyroiditis may have occurred during a previous acute infection, and that an atrophy of the gland has subsequently taken place.

Anatomy.—The shape of the skull varies largely among the mentally defective. It may be too long or too broad, or too short or too narrow, or otherwise different from the normal. Microcephaly, or a general diminution in size, is common. In hydrocephaly the skull is too big, and sometimes gigantic, while the face is of about the normal size. In a few very exceptional cases the brain is of larger dimensions and greater weight than normal, but in a large majority of instances it is smaller and lighter. Occasionally a part of the brain may be wanting, or one side may be smaller than the other. In hydrocephalus the ventricles of the brain are dilated, while the convolutions may or may not be atrophied. The convolutions are often found to be smaller, while the sulci between them are deeper and broader than normal. The grey matter of the brain may be softer or harder than the natural, or may have undergone cystic degeneration. Histologically there is found among the mentally defective a quantitative diminution of

the cells of the grey matter, a less complicated arrangement of the cells, and an increase of neuroglia. In cretins, in addition to the diminished size and weight of the brain, there is a general increase of the subcutaneous connective tissue together with atrophy of the thyroid.

Symptoms.—Mental defectives of the lowest grade exhibit no manifestations of mind. The special senses, whose organs are not uncommonly wanting, may convey sensations, but these do not arouse any associations and are not converted into perceptions. Attraction and aversion are not excited, and consequently there is no endeavour to seek the pleasurable or to avoid the painful. Food is swallowed when it is introduced into the mouth. The bladder and the rectum are emptied when they reach a certain degree of distension. Meaningless movements and noises are continuously made while the patient is awake. The face is without expression, and various deformities, such as cleft palate and hare-lip, are often present. In a rather higher grade, patients may be taught to control their sphincters, and perhaps to feed themselves and to walk. From these extreme degrees there is a very gradual transition through various grades of imbecility to a feeble-mindedness which is but little removed from the normal. Some patients can be taught to be quiet and orderly, and to play simple games or to amuse themselves, to be tidy, to wash themselves, to dress themselves, and to feed with decency. Others of yet higher capacities may be taught such occupations as laundry work, basket-making, boot-making, and carpentry.

There are a few pronounced types of mental defectives distinguished rather by physical than by mental characteristics to which reference may be made. In the *Mongolian type* of idiot the facial aspect suggests the appearance of one of Mongol race, hence the name. The palpebral aperture is almond-shaped, its long axis is obliquely placed, and the epicanthic fold is well developed. The bridge of the nose is flat. The mouth has a small aperture, but the lips are large (see Fig. 15, Vol. I, p. 305). In the case of *cretins* (see Fig. 14, Vol. I, p. 304) the face is flat in appearance, the eyelids are puffy, and the lips thick. The skin and hair are harsh and dry. The extremities are cold and purple. The pulse is slow. In *amaurotic family idiocy* the symptoms are not noticeable till towards the end of the first year. Such mental power as has by then been developed appears to pass

away. Blindness slowly comes on, the optic disc shows signs of atrophy, and the fovea a red tinge. The *microcephalic* and *hydrocephalic* types are sufficiently distinguished by the size of the head.

Treatment.—The medical treatment of cases of mental defect other than cretinism (q.v.) resolves itself into attention to the general health of the patients. Physically such patients are delicate, and proper precautions must be taken to guard them against the effects of cold and against exposure to infections. The treatment of the mental defect consists in educative efforts directed towards making use of such facilities as are present. The lowest-grade idiots are not educable in any way, and it is not possible to teach them to sit, to stand, to walk, or to be clean. Those on a slightly higher plane may be taught these elementary functions. As the scale is gradually ascended, it is found that more and more complicated acts can be taught by appropriate methods and the expenditure of much time and patient effort. Such pedagogical treatment or education can best be carried out at special schools and institutions by those who have been trained for the work.

MORAL IMBECILITY.—The conduct of the insane is often eccentric and unconventional, and occasionally improper or even criminal. Sometimes the patient has the delusion that he is being annoyed by some one person or set of persons, and, taking his protection into his own hands, assaults and possibly kills a quite innocent person. Or, as in confusional insanity, the patient, suffering from terrifying hallucinations and not recognizing his surroundings, blindly hits out in his anxiety to escape his horrible environment. Or again, as in epilepsy, criminal acts may be committed automatically and without the knowledge of the patient. Such acts performed under such circumstances are not those of moral imbecility. In this variety of mental defect the patient knows the character of his acts, is fully able to appreciate their probable consequences, and does not suffer from hallucinations, delusions, or confusion. As the education of the moral imbecile proceeds it becomes increasingly obvious that the usual effects of injunctions and punishments as observed in the normal child are not in his case produced. The moral imbecile does whatever he has a mind to do, when he likes and as he likes, and cannot be taught that certain acts are right and others wrong. He cannot be made to do

things which are at all disagreeable, and is indifferent to the feelings of others and callous to their sufferings. He is perhaps cruel to animals and to children weaker than himself, lies with effrontery, pilfers, and is sullen and bad-tempered.

As he grows older the peccadilloes of childhood and boyhood develop into the more serious offences for which greater capacity and opportunity afford scope. Idleness, thieving, gambling, alcoholism, and sexual excess comprise the patient's activities. Punishment has but little effect, and merely serves to make him more astute in avoiding it in the future. The level of the other faculties varies very much in these cases. Sometimes it is distinctly below the normal, but often it is up to the normal and may even be above it. The difficulty of dealing with such patients is very considerable. Some early fall into the hands of the police and pass into the category of the habitual criminal. Others live as a constant worry to their families and a drain upon their resources. If left to themselves, such persons sooner or later go upon the streets, or to the workhouse or the prison. They will not voluntarily submit to care except for so long as it suits them, and it is by no means always easy to certify them. There can, however, scarcely be a doubt that efficient seclusion is the only means whereby a moral imbecile can be saved from himself and society can be rid of him.

A prolonged and as thoroughly analytic an examination of the patient's mind as may be possible should be carried out, and an attempt made to discover the cause of the absence or displacement of those affects or emotional states which are normally associated with acts of right or wrong conduct, and which in the patient are plainly perverted.

E. D. MACNAMARA.

ILLUSIONS.—An illusion may be defined as a misinterpreted sensation or, more fully, as a percept experienced when an actual peripheral stimulus is present, but not the peripheral stimulus which should normally give rise to the percept in question. If, for example, a patient perceives the sound of water running from a tap as a roar of voices in the street, he is suffering from an illusion.

Illusions may occur under normal conditions, as, for instance, when a coat hanging on the door is mistaken for a human figure. They are a prominent feature in many mental dis-

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orders, particularly in febrile and other deliria. They are technically distinguished from hallucinations, in which perceptions are experienced in the entire absence of any peripheral stimulus. The distinction has, however, but little practical importance, and in all essential respects the two processes are identical. (*See HALLUCINATIONS.*)

BERNARD HART.

IMBECILITY (*see* IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, MORAL IMBECILITY).

IMMUNITY.—The term immunity, in its usual application, signifies the power of the animal body to resist infection with parasitic organisms or the injurious effects of their products. From the biological standpoint, immunity represents a natural adaptation on the part of the body to protect itself from the aggression of other living organisms. The animal organism is thus provided with a natural immunity mechanism which affords it some relative protection from the invasion of micro-organisms, and the process of natural recovery from infective disease signifies the further adaptation of this mechanism to effect either the destruction of the particular infecting organism or the elimination of its toxic products.

Immunity is strictly relative, and the protection resulting from an immune state depends on the degree of immunity and the number and virulence of the organisms which gain access to the body; a low degree of immunity may confer protection against a small number of organisms or against organisms of low virulence, while even a high degree of resistance may not be sufficient protection from a large "dose" of organisms or from organisms of exalted virulence.

Immunity to a particular infection may be *natural* to the species or the individual. It is well known that various animals are immune to certain pathogenic organisms to which other species are highly susceptible, and during an epidemic many individuals fail to contract the disease even after exposure, or suffer only slightly, while others succumb. Immunity may be *acquired*, as in the course of an infective disease, and the development of special resisting powers against the causative organism may be the deciding factor in the recovery of the patient from the infection. This form of immunity may persist for long periods after convalescence, and even throughout life, as in the case of smallpox. On the other hand,

it may be of short duration, e.g. pneumococic and streptococic infections.

An acquired immunity is also *specific* for the particular virus, and is essentially the result of a reaction on the part of the tissues to the particular organism or its products; it can be produced artificially by inoculating an animal with the virus so modified that it is unable to reproduce the actual disease though still capable of bringing about the immunity reaction. In this case the immunity is designated *artificial*.

The blood-serum of an animal which has been "actively" immunized, when injected into the body of another animal, may render the latter temporarily immune in the same specific manner. Immunity conferred in this way is termed *passive*.

Though artificial immunity was first demonstrated experimentally in the case of smallpox by Jenner before it was known what part micro-organisms played in infective disease, it has only been as the result of experimental investigation of pathogenic organisms that the principles of immunity have come to be definitely understood and generally applied in the prophylaxis and treatment of disease. Thus, investigation of the serums of immune animals led to the discovery of antitoxins and bacteriolysins, and showed that the immunity of an animal depended largely on certain properties of its serum. It was also shown how the phagocytes of the body play an important part in natural immunity and constantly help to defend the tissues from invading organisms. The local inflammatory reaction which follows certain infections represents a defensive reaction of the tissues in which the phagocytic action of the leucocytes is an important factor.

An **active immunity** may be artificially effected by—(1) the introduction into the body of organisms of attenuated virulence or in non-fatal doses; (2) injecting their toxins in non-fatal doses; (3) injecting killed cultures; (4) introducing the organisms by a route other than the natural avenue of infection, e.g. cholera vibrios injected subcutaneously are killed locally and do not cause a choleraic condition; (5) injecting with the organisms some substance which modifies their action. This immunity can be exalted to a high degree by progressively increasing doses, and as the immunity develops in the course of the immunizing process the animal will ultimately tolerate doses which would otherwise be fatal.

The **virulence** of organisms can be **attenuated**

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for purposes of immunization in various ways: (1) cultivation for a time on an artificial culture medium; (2) cultivation in a current of air; (3) "passage" through a series of animals of another species—the smallpox virus after passage through the calf becomes so modified that it fails to produce the characteristic disease in man, though still capable on inoculation of bringing about a state of immunity to smallpox; (4) cultivation in the presence of a weak concentration of antiseptic; (5) cultivation in an atmosphere of compressed air; (6) cultivation at high temperatures—Pasteur's anthrax vaccine consisted of cultures of *B. anthracis* grown at 42° C.; (7) drying in air—the hydrophobia vaccine (Pasteur) is prepared from the spinal cords of infected animals dried in air for definite periods.

In the case of an organism which produces a soluble toxin (exotoxin) the filtrate of fluid cultures which have been passed through a porcelain filter constitutes a sterile toxic fluid, and can be injected in sublethal doses to produce a state of immunity without affecting the health of the animal.

Killed cultures (vaccines) are frequently employed for artificial immunization both in man and in animals, e.g. typhoid and cholera vaccines, and heat-killed cultures were originally used by Wright in the treatment of bacterial infections, the vaccine being employed to produce an immunity to the infecting organism. The immunizing properties of dead cultures are hardly comparable with the effect of living organisms, e.g. in the case of *B. pestis* and streptococci, and the sterilization of the vaccine may cause some deterioration of the active constituents of the organism. In the preparation of vaccines it is therefore advisable to kill at as low a temperature as possible. Bail has shown that certain organisms produce a special toxic substance (*aggressin*) when actually growing in the tissues and fluids of the body, and the development of a special *anti-aggressin* has been demonstrated. The multiplication of organisms in the tissues may be necessary, therefore, to stimulate a powerful immunity reaction. *Sensitized vaccines* (Bes-redka) are cultures which have been treated with a specific antiserum; they are generally less toxic than the untreated vaccine, and it is claimed for them that the resulting immunity is more rapidly established and of longer duration. They are especially applicable in the case of vaccines of which even small doses produce a profound toxic effect, e.g. vaccines of

B. dysenteriae (Shiga) and of the gonococcus. *Detoxicated vaccines* (Thomson) are cultures from which the bacterial toxin has been removed; large doses can be given without toxic effects.

Passive immunity.—The serum of an immune animal is termed an *immune serum* or *antiserum*, and the specific immune elements present in the serum are described as *anti-substances* or *antibodies*. Passive immunity depends on the transference of these constituents from the immunized animal to the otherwise non-immune animal. An antiserum which acts by neutralizing the toxins of the organism is called an *antitoxic serum*, and the active constituent is termed *antitoxin*. On the other hand, an immune serum may affect the organism directly, and is then designated an *antibacterial serum*.

Antigens.—The substances in the organism which give rise to the specific antistances are called *antigens*, and the same term would be applicable to the exotoxin used for immunization.

Apart from bacterial immunity, other cells and substances act as antigens and on injection into animals stimulate the production of specific antistances, e.g. red blood-corpuscles and other animal cells, ferments, serum, toxic vegetable proteins (ricin, etc.), animal venoms, and in general all proteins alien to the animal injected. Thus the red corpuscles of the ox injected into the rabbit lead to the development in the rabbit's serum of antistances (*haemolysins*) which are specific for the corpuscles of the ox, and under certain circumstances lead to hæmolysis of ox-blood. In the same way an animal immunized with cobra venom reacts by producing an *antivenin* which is present in its serum and neutralizes the venom, just as antitoxin acts on toxin.

To produce immunity, the *antigenic substance* must be introduced into the tissues, i.e. must be given parenterally. Generally, the immunizing substance, if administered by feeding, is deprived of its foreign specificity in the alimentary tract (see below).

In general, only proteins and closely allied bodies are capable of acting as antigens, and an animal can only be immunized against antigenic substances which are foreign to its own tissues. Evidence that other substances (e.g. lipoids) can act as antigens is so far inconclusive. It has been shown, however, that the injection of certain simple chemical substances (e.g. saccharose) leads to the development in

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the blood of an antagonistic ferment which acts by decomposing the substrate (Abderhalden and Weinland). This condition is analogous, in some respects, to immunity against proteid substances.

Antibodies usually appear in the serum in appreciable amount after five days, but it is probable that small though effective amounts may appear much earlier.

The **specificity of antibodies** for the particular antigen is usually well marked, though specific bacterial antibodies may also act to some extent on biologically allied organisms (*group reaction*). As a rule, specificity is manifested for the biological species, but may even be more restricted, as in the case of the *B. coli* group, where certain antibodies are specific for the individual strain (Mackie). **Hetero-genetic antibodies** have been described—i.e. antibodies which are specific for an antigen which has no biological relationship to that used for immunization. Thus the injection of guinea-pig's kidney tissue into a rabbit leads to the development of a powerful antibody to sheep's red corpuscles; in typhus fever the serum may show an immunity reaction (agglutination) with *B. typhosus*, which has no etiological relationship to this disease. In typhus fever also the serum may agglutinate an organism of the *B. proteus* group (Weil-Felix reaction). The explanation of these phenomena is still obscure, but it is known that specific antibodies may occur naturally in the serums of animals apart from any reaction to antigen. Thus an antibody (hæmolysin) to the red corpuscles of the ox is very frequently present in the serum of the guinea-pig.

Specificity is closely related to the chemical constitution of the antigen, and chemical changes (e.g. serum-protein treated with nitric acid) render the altered antigen specifically different as regards the antibodies produced by it, when compared with the original antigen (Obermeyer and Pick, and others).

Antitoxic serums.—One of the best examples of an antitoxic serum is the *diphtheria antitoxin*, the serum of a horse which has been treated with successive, increasing doses of diphtheria toxin (the filtrate of a fluid culture of *B. diphtheriæ*). If appropriate amounts of this antitoxic serum and toxin are mixed and injected into a susceptible animal, no toxic effects result; normal serum, however, is incapable of neutralizing the toxin and preventing its lethal effect. It is apparent that the tissues are responsible for the production of

the antistubstance, for the immunized animal, after several bleedings representing altogether its original blood volume, may still show the same content of antitoxin in its blood as was originally present.

Antitoxin exhibits a marked affinity for toxin, and unites with it to form a non-toxic compound; this combination may occur *in vitro*, and represents a firm union, though toxin can be dissociated and recovered from the toxin-antitoxin combination by the action of an acid. Toxin is therefore not destroyed by the antitoxin. The combination takes a certain length of time to occur, and is analogous to a chemical reaction; it occurs more rapidly in strong solutions, and is also accelerated by warmth. The neutralization of toxin by antitoxin is complex, and not similar to the reaction between a strong acid and alkali. Ehrlich found that if the largest amount of toxin which could be neutralized by a certain fixed amount of antitoxin was estimated (L_0) and larger amounts of toxin were then added until a lethal effect was produced (L_+), i.e. when one minimum lethal dose was left free in the mixture, the difference between the L_+ and L_0 doses was invariably more than a minimum lethal dose (*Ehrlich phenomenon*). It has been shown that an analogous condition exists in the case of certain colloidal reactions, e.g. the neutralization of arsenic trioxide by ferric hydroxide (Biltz). The amount of toxin neutralized by antitoxin is greater when the toxin is added at once than when it is added in separate fractions (*Danysz phenomenon*); this phenomenon has also an analogy in the interaction of two colloid substances. Toxin and antitoxin are apparently of colloidal nature, and the reaction between them may therefore be of the nature of a colloidal reaction. Ehrlich suggested that the apparently complex nature of the interaction was due to the development of *toxoids* from the toxin molecule, which are capable of combining with the antitoxin, though devoid of poisonous effects. According to Ehrlich, the toxin molecule consists of a haptophore group which unites with certain cell molecules, and a toxophore group which acts after combination with the cell and produces the toxic effects. In this way the latent period following the injection of the toxin was explained, and toxoid represented the toxin molecule deprived of the effective action of its toxophore group, but with the haptophore group intact. In Ehrlich's conception of antitoxin development in the immune animal, the

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toxin combines firmly with certain cell "receptors" (side chains of the protoplasm molecule); these are accordingly replaced by reproduction of similar receptors, which are ultimately produced in excess as the result of successive doses of toxin, and are set free in the blood-stream as the antitoxin of the immune serum. Toxoid is capable of inciting the production of antitoxin, and this supports the assumption that the haptophore group combines with the cell and stimulates the production of antitoxin.

As to the actual **chemical structure of toxin**, little is known. Toxins have never been prepared as chemically pure substances, and are invariably associated with proteins. They bear some similarity to enzymes, and are probably of colloid nature. Similarly, antitoxins have never been isolated from the serum proteins, though analysis of serum shows that they are associated with the globulins.

The neutralizing effect of antitoxin occurs also *in vivo*, and thus antitoxin can be introduced into the body both for prophylactic and for therapeutic purposes.

An antitoxic serum requires careful **standardization**, and this is carried out in the case of diphtheria antitoxin by Ehrlich's method. The so-called "immunity unit" was the amount of serum which neutralized 100 minimum lethal doses of toxin for a guinea-pig of 250 gm. weight. Thus, for estimating the antitoxin content of a serum, the strength of toxin must be determined. Toxins are, however, unstable, and in practice it is usual to employ a standard antitoxin (preserved *in vacuo* in the dry condition) for quantitative comparison with the unknown serum as regards its power of neutralizing toxin.

Concentrated antitoxin.—The antitoxin, being associated with the serum-globulins, can be easily concentrated by precipitating and separating the globulins in 50-per-cent. saturated solution of ammonium sulphate, dialysing to remove the salt, and then redissolving in isotonic sodium chloride. In this way a concentration of six times may be obtained (Park), and the use of these serums enables larger doses to be given conveniently and with less tendency to serum sickness (*see p. 76*).

Antibacterial serums.—A powerful antibacterial serum may be obtained by immunizing an animal in the first place with successive injections of dead cultures and, when sufficient immunity has been established in this way, with doses of living organisms, and ultimately

with injections of living cultures of exalted virulence. The immune serum is protective against the particular organism, though not in virtue of antitoxic properties. By injecting the serum into a non-immune animal, passive immunity can be produced. These serums have been extensively used in the treatment of acute infections—e.g. streptococcic, meningococcic, pneumococcic infections, etc.

Antibacterial serums may act on organisms in various ways. (1) The organism may be dissolved (*bacteriolysis*) and killed (*bactericidal action*); (2) the organism may be rendered more susceptible to phagocytosis by body cells (*opsonic, bacteriotropic action*); (3) *agglutination* or clumping of suspensions of the organism *in vitro* may be produced; and (4) *precipitation* of its soluble products.

These properties may all be exhibited by the serum of individuals infected with certain pathogenic bacteria—e.g. *B. typhosus*, *V. cholerae*; the effects are highly specific for the particular bacterial species.

Normal serum may also display a certain degree of bactericidal and opsonic action in a relatively non-specific manner, a mechanism which is an important factor in the natural immunity of the animal body. The bactericidal action of normal serum is due to a normal thermolabile constituent of all animal serum—*complement, cytase, alexin, addiment*—and its action is non-specific. Normal serum also acts on organisms so as to render them susceptible to ingestion by leucocytes. This effect is relatively non-specific, and, like bactericidal action, depends on a thermolabile complement-like body in the serum (*normal opsonin*). As originally shown by Wright and Douglas, leucocytes washed free of serum have no power of ingesting staphylococci; in the presence of fresh serum phagocytosis occurs, while heated serum (60° C.) is inactive; if the cocci are treated with the fresh serum and then freed from serum by washing, they are ingested by the leucocytes, showing that the effect is on the organisms and not the leucocytes.

Bactericidal action by an immune serum is well exemplified by *Pfeiffer's reaction*. *V. cholerae* and a specific immune serum are injected together into the peritoneum of a guinea-pig; after a time, if the peritoneal fluid is withdrawn, it is found that the organisms are undergoing a process of lysis and disintegration. The same phenomenon can be demonstrated *in vitro* at 37° C, and is independent of the body-cells. Non-immune serums may

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produce this effect to a limited extent, but exceedingly minute quantities of an immune serum may exhibit bacteriolytic action, and the effect is specific for the particular organism. Immune serum heated at 57° C. is deprived of bactericidal action *in vitro*, but an amount of fresh serum which has no effect by itself at once restores the activity of the heated serum. Bactericidal action by an immune serum is thus due to the combined action of a thermostable specific immune substance along with the normal thermolabile complement. In Pfeiffer's *in vivo* experiment the immune substance is introduced along with the organisms, and the animal's complement reacts with it to produce bacteriolysis.

An analogous phenomenon is the hæmolytic action of the serum of an animal which has been immunized with the red blood-corpuscles of another species; a thermostable immune body is developed which has no action by itself, but along with complement produces lysis or laking of the homologous corpuscles. This phenomenon can be easily studied in test-tube experiment, and in this way valuable information regarding the mechanism of immune body and complement action has been obtained.

Bacteriolysis and hæmolytic action are due, therefore, to (1) a specific thermostable immune body (*amboceptor*, *fixateur*, *substance sensibilisatrice*), which may be developed by the parenteral injection of the antigen, (2) the thermolabile complement which is present normally in the serum, and does not undergo any increase during immunization. Red corpuscles and bacteria combine with the immune body and are then "sensitized"; if washed free of serum they can still be lysed on the addition of complement. Complement does not unite directly with red corpuscles, but is firmly combined (absorbed, deviated) by the corpuscles along with the immune body. The immune body, therefore, seems to act as a combining link between the antigen and complement (hence the term *amboceptor*). Sensitized red corpuscles may take up more complement than is required for the lytic effect. It would appear that the amboceptor in an immune bacteriolytic serum acts by bringing into combination with the cell a larger amount of bactericidal complement than would act under ordinary conditions (Muir).

The chemical nature of immune body and complement is obscure, and they are indissociable from the serum proteins. Complement is of complex constitution, and has been shown to represent several different components; and

though whole complement is extremely thermolabile, a thermostable constituent has been demonstrated. It has been suggested that complement is of enzyme nature, but, unlike ferments, it is used up quantitatively in the process of hæmolysis or bacteriolysis. Attempts have been made to explain hæmolysis by an immune serum on a physico-chemical basis. It has been found that colloidal silicic acid could replace a hæmolytic immune body and produce hæmolysis along with complement (Landsteiner and Jagic), but thermostable constituents of serum are also capable of effecting hæmolysis along with certain colloids—e.g. brilliant-green and other colloidal triphenyl-methane compounds (Mackie).

Antibody reactions are often marked by "zone phenomena" (a feature also of colloidal reactions), in which the particular effect is only produced when the reacting components are present together in definite relative proportions (e.g. *Neisser-Wechsberg phenomenon*—bacteriolysis inhibited by excess of immune body with a given quantity of complement). Such observations suggest that these reactions are of the nature of colloid phenomena, and the physico-chemical aspect of immunity phenomena generally presents an interesting subject for further investigation.

Bactericidal action is well marked in the case of typhoid and cholera immune serums, but certain antibacterial serums may show little or no bactericidal effect, e.g. streptococcus, staphylococcus, plague antisera.

Immune opsonins.—Immune serums may contain, in addition to the normal non-specific opsonin, an immune specific opsonin which, unlike the normal opsonin, is definitely thermostable (Muir and Martin), and effects a much greater opsonic action on the particular organism. Certain immune serums probably owe their action for the most part to this body, and, in the case of certain infections occurring naturally, a similar immune substance is developed and appears in the blood.

The activity of the serum opsonins in various infections has been taken as some indication (*opsonic index*) of the resisting power of the individual, and methods have been employed for estimating opsonic effects.

As regards the actual effect of opsonins on bacteria we have practically no knowledge, though it is known that the viability of the organism is not interfered with. It has been suggested that they act by neutralizing agglutinin, which is specially toxic to leucocytes.

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Agglutination.—The power or agglutinating into clumps the homologous organism is also one of the properties exhibited by an antibacterial serum, e.g. serum of a man or animal infected naturally (Gruber-Durham or Widal reaction in typhoid fever), or immunized artificially with a particular organism. This effect is regarded as due to actual substances in the serum (*agglutinins*) which are generally specific though often capable of acting on organisms allied to the homologous species (*group reaction, coagglutination*). The degree of development of agglutinins in disease and in artificial immunity is extremely variable. The part played by these bodies in actual immunity is doubtful, and it is questionable if agglutination occurs to any extent *in vivo*. Agglutinins do not interfere with the viability of the organism. They are relatively thermostable. Their action does not depend on the vitality of the organism, e.g. dead bacteria are also susceptible. The agglutinin combines with the organism, and is used up quantitatively in producing its effect.

The mechanism of agglutination is not yet fully understood, though various physico-chemical explanations of the reaction have been suggested. Zone phenomena may be exhibited with relative excess of agglutinin. Salt is necessary for the reaction, and agglutinins will not affect organisms suspended in distilled water; agglutination has been compared with the precipitation of colloidal suspensions of clay by salt. In this case it would be necessary to presuppose that the bacterial envelope acts as a protective colloid, and that the agglutinin in some way interferes with it and exposes the organism to the action of the salt.

The red corpuscles of one individual may be agglutinated and lysed by the serum of others of the same species (*isohæmagglutinins* and *isolyins*), and on this depend injurious effects following blood transfusions in man. Hence the necessity for ascertaining the effect of the recipient's serum on the cells of the donor, before carrying out a transfusion.

Precipitins are also characteristic constituents of antibacterial serums, and produce a precipitate in filtered cultures of the homologous organism—i.e. the soluble products of the organism. The actual precipitate, however, is derived mainly from the antiserum. Precipitins are probably bodies closely allied to agglutinins, and have certain similar characters.

Serum-precipitins.—The serum of one animal injected into another species also leads to the

development of a specific precipitin. In this way the presence of minute amounts of a particular serum can be determined by means of an homologous antiserum.

Deviation of complement.—Just as bacteria along with a specific bactericidal immune body absorb complement, so also, apart from bactericidal action, immune serums generally may contain antibodies which, along with the antigen, produce a marked absorption or deviation of complement. The reaction can be shown in the following way: Antigen and antiserum are mixed, and complement (e.g. fresh guinea-pig's serum) is added; after incubation for a time a blood suspension sensitized by the addition of an homologous immune body is added. If the complement has been deviated, it will not be available to act on the sensitized corpuscles, and hæmolytic does not occur. The Wassermann syphilis reaction is carried out in a similar way, but the antigen (a lipid emulsion) has apparently no relationship to the causative organism of the disease, and the phenomenon can hardly be regarded as an immunity reaction.

Site of formation of antibodies.—While all body cells may be capable of producing antibodies, and local immunity has been demonstrated in animals (e.g. the immunity of one eye inoculated with smallpox vaccine, the other eye still remaining susceptible), it is likely that the leucocyte-forming organs—spleen, lymph-glands and bone-marrow—and the vascular endothelium are chiefly responsible for the production of immune substances.

Natural immunity.—On the natural-immunity mechanism of the body the animal depends for the relative natural resistance it possesses to the invasion of micro-organisms generally: (1) phagocytic action of leucocytes and certain other cells, (2) the normal non-specific opsonin, (3) bactericidal complement.

This natural resistance is subject to considerable variation, and depends to some extent on the general vitality of the body and the health of the tissues. Any factor which tends to depress the general health or damages the tissues and interferes with their functions will render the individual more susceptible to infection. It is well known that starvation, cold, fatigue, and mechanical damage to tissues may predispose to infection, and one infection may predispose to another in the same way (secondary infection).

The different animal species show great variation in their susceptibilities to different

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infecting organisms, e.g. fowls are insusceptible to tetanus, and white rats are immune to *B. anthracis*. While this specific natural immunity may be due to a high degree of phagocytic action by the leucocytes towards the particular organism (e.g. *B. anthracis* in the tissues of a white rat), or specific bactericidal action by the serum, insusceptibility of the tissues to the bacterial toxin is also an important factor in natural immunity. Individuals, too, vary considerably as regards their power of resistance, and in man susceptibility, apart from other factors, varies with age.

It has also been suggested that natural immunity is due to the absence from the body of some necessary pabulum for the particular organism (*athrepsy*). It would be difficult, however, in this way to explain the nonsusceptibility of certain animals to organisms which show little restriction as regards growth on artificial media.

Recovery from infection.—The invasion of the body by organisms is followed by reactionary changes on the part of the tissues. The process of inflammation is one of these, and is characterized by the emigration of leucocytes from the capillaries at the focus of infection. Their obvious rôle is the ingestion and intracellular destruction of the invading organisms, though in some instances ingestion does not affect the viability of organisms—e.g. gonococci, meningococci in leucocytes of exudate. In the higher animals phagocytic body cells destroy bacteria in the same way as similar cells in certain minute animals (e.g. daphnia) ingest parasitic yeasts which if left free would cause a fatal disease (Metchnikoff). Besides the leucocytes, endothelial cells and probably embryonic connective-tissue cells act as phagocytes. In many infections also there is an active leucocytosis, and large numbers of leucocytes are mobilized, as it were, for phagocytic action as the result of increased activity of leucocyte-producing tissues.

The attraction of leucocytes to the focus of infection may be regarded as a chemiotactic one (Leber) depending on a certain chemical substance in the infecting organisms. It has been supposed that chemiotactic influences depend on alterations in surface tension; diminution of surface tension leads to movement of an amoeboid cell in the direction of diminished tension, and if organisms produce diffusible substances which lower tension, leucocytes would thus be attracted to the focus of infection. The action of leucocytes depends on

the serum-opsonins, and the development of an immune specific opsonin increases the phagocytosis of the particular organism. At the same time, the bacterial toxin, by damaging or destroying leucocytes, may counteract this protective effect. Antitoxins and bactericidal immune bodies developed in response to the infection will also determine the effective resistance of the body and recovery from the disease.

It is known that individuals exposed to infection may never show any appreciable clinical signs of infection and yet contain specific immune substances in their serum, e.g. agglutinins in the serum of persons exposed to *B. typhosus*, antitoxin in the serum of healthy diphtheria contacts. It is probable in such cases that the organism has actually gained access to the tissues, but has been quickly destroyed. In those infections in which relapses occur the immunity has been a non-sterilizing one and short-lived. In various conditions, also, recovery does not necessarily signify complete sterilization, and the organisms may persist in some situation where they are protected from the action of leucocytes and serum antibodies, e.g. typhoid bacilli in the gall-bladder. According to Welch, an organism may actually immunize itself against the antibodies of the host. In this way a chronic infection may become established, e.g. chronic protozoal infections.

Persistence of immunity.—Even after immune substances have apparently disappeared from the blood of previously immune individuals, the power of resisting infection may still be greater than normal owing to the fact that the body cells which have previously responded may more readily react subsequently to an immunizing influence and quickly produce antibodies to deal with the infection.

A person once inoculated with *B. typhosus* vaccine, on contracting a paratyphoid infection after a period when typhoid agglutinins are only present in minimal amounts, may again develop high-titre agglutinins to *B. typhosus* (Mackie and Wiltshire). This shows a ready tendency on the part of the tissues which have once produced specific antibodies to develop similar antibodies again in response to a suitable stimulus, for of course in the uninoculated individual a paratyphoid infection would not lead to the development of independent high-titre agglutinins for *B. typhosus*.

Metchnikoff's theory of acquired immunity.—Metchnikoff based his explanation of

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immunity entirely on the phagocytic action of body-cells and their effect in destroying and digesting organisms by intracellular enzymes; thus the susceptibility or resistance to infection was supposed to depend on the activity of the phagocytes: intracellular ferments (cytases) were set free when the phagocyte was injured (phagolysis), e.g. on withdrawing blood from the body, and were then present in the serum in the free state. The liberated cytase represented the free complement. Macrocytase and microcytase were distinguished, the former derived from macrophages (e.g. endothelial cells) and acting on protozoal organisms, and the latter from the microphages (polymorphonuclear leucocytes) and active against bacteria. Immune bodies were regarded as in the nature of "auxiliary ferments" (like enterokinase).

This theory hardly represents a complete biological explanation of immunity, though the fundamental fact—the phagocytic action of body-cells—is undoubted. It in no way explains the specificity of different immune bodies, and there is evidence to show that free complement is present in the plasma. Opsonic action also shows that the phagocyte is not independent of the serum.

Ehrlich's theory.—Ehrlich supposed that the protoplasm molecule nourished itself by "side-chains" (receptors) having special affinities for different protein substances, and that the protein antigen, combining with the corresponding receptor and throwing it out of action for the purpose of the cell, led to an excess production of new receptors, which were shed into the blood-stream and represented the specific antibody. The different types of antibodies were referable to the different "orders" of receptors and their action on the food molecule—(1) those which merely combine with the food molecule or antigen (e.g. antitoxin and antiferment); (2) those which combine with the food molecule or antigen and also produce some change in it (e.g. agglutinin, precipitin); (3) those which combine both with the food molecule and with complement so that the ferment-like complement can act on the food material and prepare it for assimilation by the cell (e.g. hæmolysin, bacteriolysin).

One of the principal difficulties in reconciling this conception with the facts of immunity is that to produce immunity the foreign protein must reach the tissue directly by parenteral introduction, while in nutrition protein material

is chemically altered in the alimentary duct and its foreign specific characters are annulled.

According to Vaughan, pathogenic organisms infect the body in virtue of possessing ferments which split up tissue protein, and so afford pabulum required for their proliferation. During this stage, which represents the incubation period, no symptoms occur, since the products of protein cleavage are at once assimilated by the bacteria. Vaughan has shown that the protein molecule contains a poisonous group, and he supposes that in the active stage of an infection, when symptoms appear, what has happened is that now the tissues have supplied a ferment which destroys the bacteria and thereby sets free the protein poison. Immunity is probably due to the presence of antiferments in the body.

Anaphylaxis.—Under certain conditions the parenteral injection of alien protein produces a specific "sensitizing" effect (hyper-sensitiveness, anaphylaxis), so that subsequent injection of the particular substance produces toxic and even fatal results. This condition is closely related to immunity, though anaphylaxis and immunity are directly opposite effects. Substances which produce immunity (antigens, bacteria, serum, etc.) may lead to anaphylaxis, and the latter state is also related to the presence of antibodies in the blood. The apparent factor that decides the occurrence of anaphylactic phenomena is the interval between the sensitizing and the subsequent injection.

The condition is exemplified by the *phenomenon of Theobald Smith*: guinea-pigs injected with even 0.01 c.c. of horse-serum, when re-injected intravenously after ten days with 0.2 c.c. of the same serum, may die within a few minutes, while a single large dose of serum produces no injurious result. The toxic effects are mainly on the nervous system. Intravenous injection of the second dose is usually more effective than subcutaneous injection, but if it is injected in small fractions anaphylactic symptoms do not occur. The result depends also on the amount of the antigen introduced into the sensitized animal.

If, after the sensitizing injection, but before the anaphylactic condition has developed, a second dose is given, the animal is protected from the effects of a subsequent dose, and an animal which has recovered from the anaphylactic shock is similarly protected against a further injection (*antianaphylaxis*).

Passive anaphylaxis can also be produced by transference of the serum of a sensitized to a

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normal animal, and the condition appears to depend on the presence of antibodies in the serum—allergen, sensibilisin, anaphylactin. Anaphylaxis has not been fully explained. The antibody reacts with the antigen (if present in certain amounts) and a highly toxic substance is produced (anaphylatoxin). Vaughan and Wheeler claim that protein injected parenterally leads to the development in the serum of a specific protease, so that the same protein when reinjected after an interval in sufficient amount is broken up with great violence and the poisonous group of the protein molecule is liberated. Vaughan has demonstrated the highly poisonous properties of "split" protein.

Anaphylaxis is of the greatest importance in relation to serum-therapy. The administration of a single dose of horse-serum (e.g. diphtheria antitoxin) frequently leads to toxic effects after 8–10 days, e.g. local inflammatory reaction at site of injection, fever, exanthemata, oedema, albuminuria, swelling of lymph-glands and joints. If a second dose of serum is given, pronounced anaphylactic effects may occur, depending on the interval. Thus, (1) if less than ten days after the first dose, no reaction occurs; (2) if twelve days to six weeks, there is an "immediate" reaction with marked symptoms, local oedema, pyrexia, skin rash, dyspnoea, etc.; (3) if over six months, an "accelerated" reaction occurs, like that produced by a first dose, but more severe and more rapidly developed; (4) between six weeks and six months both the immediate and accelerated reactions may occur. The immediate reaction corresponds to experimental serum anaphylaxis in animals, but if the second dose is administered subcutaneously the toxic effects are very rarely fatal in man. On the other hand, if the administration is intravenous or intraspinal, dangerous results may ensue. In this case the attempt should be made to establish antianaphylaxis by *Besredka's method*. (1) Where treatment is not very urgent, inject 10–20 c.c. of serum subcutaneously; after an interval of four to twenty-four hours a full dose of serum may be given intraspinally; (2) 2 c.c. of serum are given intraspinally, and then, after an interval of at least two hours, the full dose may be administered; (3) where an intraspinal or intravenous injection of serum is a matter of urgency, dilute 5 c.c. of serum with 10 volumes of 0.85-per-cent. sodium chloride solution and inject 1 c.c. of the mixture into a vein, this being

succeeded at intervals by further injections—after four minutes 3 c.c., two minutes later 10 c.c., again two minutes later 25 c.c.; ten to fifteen minutes after the last injection the full therapeutic dose may be administered intravenously or intraspinally.

Hypersensitiveness can, if necessary, be tested for by injecting 0.25 c.c. of horse-serum intradermically. The development of an urticarial patch within thirty minutes, followed by the formation of a vesicle and a surrounding erythematous area, constitutes a positive reaction. The absence of a reaction within forty minutes represents a negative result.

When prophylactic doses of diphtheria antitoxin are given to children who might later develop the disease and require full serum-treatment, an antitoxic serum from cattle may be used, so that later the usual horse-antitoxin may be given without danger. In vaccine-treatment anaphylactic effects have not been usually observed.

Cutaneous hypersensitiveness to the products of the causative organism occurs in various infections (*allergic reaction*)—e.g. tuberculin in tuberculosis, luetin in syphilis, typhoidin in typhoid fever—and is analogous to other anaphylactic phenomena. These reactions have been used as diagnostic tests.

Hay fever has been ascribed to a natural sensitiveness to certain pollens, and individuals may even exhibit anaphylaxis to particular proteins absorbed from the alimentary tract, e.g. in the case of milk, eggs, etc. To this abnormal sensitiveness to foreign proteins Freeman has given the name of *toxic idiopathy*.

APPLIED IMMUNITY: (1) ACTIVE IMMUNIZATION (VACCINE-THERAPY)

Active immunization may be employed both prophylactically and therapeutically. For this purpose vaccines representing the causative organisms have been employed. Living viruses of attenuated virulence have been used in the case of smallpox and rabies vaccination. The Jennerian vaccine (cowpox lymph) may be regarded as the smallpox virus attenuated by passage through calves (*see ante*, p. 65). Pasteur's process of rabies vaccination consists of injecting in successive doses emulsions of spinal cords of infected rabbits containing the living virus. The virus is attenuated by drying the cords for varying periods in air. The injections are graded so that the first doses consist of attenuated material, while the final doses represent a fully active virus.

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Dead cultures have been commonly used for active immunization where the causative organism has been ascertained and cultivated artificially, e.g. *B. typhosus*, *B. paratyphosus-A* and *-B*, pneumococci, *B. pestis*, *V. cholerae*, etc.

Preparation of killed-culture vaccines.

—An 18–24 hours' culture on solid media is emulsified in 0.85-per-cent. salt solution so as to yield a concentrated suspension, and is sterilized, usually by heat, at as low a temperature as possible (56°–60° C. for one hour). Excessive heating may lessen the antigenic properties of the vaccine. Sterility is then determined by making inoculations from the emulsion on solid medium, which is incubated for forty-eight hours. The vaccine is standardized according to the number of million organisms per c.c. It is for this reason that young cultures are employed, as in older cultures a proportion of the bacteria undergo autolysis and a numerical estimation would not indicate the actual concentration.

Vaccines may be **standardized** in various ways: (1) By making a 1-in-40 dilution of the emulsion in salt solution with sufficient methylene-blue to stain the organisms; a drop of the mixture is mounted on a hæmocytometer counting stage, and, after it has stood for a time to allow the bacteria to settle, a count is made and the estimation of the number of organisms per c.c. can be arrived at as in the case of blood-counts. (2) By mixing blood and vaccine in definite proportions and making from the mixture films, which are stained with Leishman's fluid. The proportion of organisms to red blood-corpuscles is estimated under the microscope, and in this way, the red cell count being known, the strength of the vaccine can be calculated (*Wright's method*). (3) By centrifugalizing the original suspension in special graduated tubes in which the bacterial sediment can be measured and then used for making a 1-per-cent. emulsion. The concentration of different organisms in standard emulsion has been carefully estimated by enumeration methods, and in this way a vaccine of a particular organism can be quickly standardized (*Hopkins's method*). Definite concentrations can then be made up from the stock emulsion (e.g. *A*, 100 million organisms per c.c., and *B*, 1,000 million organisms per c.c.) and placed in vaccine bottles or sealed ampoules (1 c.c. capacity); the requisite doses are obtained by taking the appropriate amounts of these concentrations. If necessary, the full contents of the various ampoules may be made to represent

particular doses by diluting appropriate amounts of the stock emulsion in a fixed volume of salt solution (1 c.c.).

Preparation of sensitized vaccines (see p. 65).—Dense emulsions in salt solution are prepared from cultures and a certain amount of a specific antiserum is added; after six hours the mixture is centrifugalized and the sediment is thoroughly washed in salt solution; the bacteria are then resuspended and heated at 60° C. for one hour to ensure sterility.

Prophylactic immunization.—Apart from smallpox and rabies, prophylactic vaccination has proved an effective means of increasing resistance to various infections, e.g. typhoid and paratyphoid fevers, pneumococcal pneumonia, plague, cholera.

Typhoid-paratyphoid vaccine.—The typhoid-paratyphoid vaccine was extensively employed during the late war with most successful results, and proved an extremely effective means of controlling enteric infections. The British Army vaccine ("T.A.B.") consists of heat-killed cultures of *B. typhosus*, *B. paratyphosus-A* and *-B*; it is injected subcutaneously in the arm near the insertion of the deltoid or in the infraclavicular region. At least two injections are required to produce such immunity as will afford efficient protection, and, if possible, a course of suitably graded injections is advisable. It is usual to inject relatively large doses, e.g. as first dose 500 million *B. typhosus*, 250 million *B. paratyphosus-A*, and 250 million *B. paratyphosus-B*, followed after seven to ten days by double this dose. The injection usually leads to local and general toxic symptoms, due to the bacterial endotoxin. The local effects consist of an inflammatory reaction with swelling and tenderness, developing about six hours after the inoculation and attaining its maximum in about twenty-four hours. The related lymph-glands are also somewhat swollen and tender, and there is frequently pyrexia (100°–103° F.), with rigors, headache, vomiting, diarrhoea, and general malaise. The reaction lasts forty-eight hours at most. To lessen the local swelling 30-gr. doses of calcium chloride may be given at the time of injection and repeated after twelve hours.

Negative phase.—According to Wright, immediately following the injection of vaccines there is a temporary lowering of resistance, as evidenced by diminution of opsonin content in the serum. It has been supposed that for a short period following typhoid vaccination there might be a negative phase during which

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the individual is more susceptible to infection. Leishman has doubted this possibility, but the writer has observed cases in which typhoid-paratyphoid vaccination appeared to predispose in this way. Such instances must, however, be regarded as exceptional.

Duration of immunity.—It has been estimated that the immunity persists for about eighteen months and then gradually declines so that reinoculation after a year is advisable. In this case one injection, corresponding to the second dose of the original vaccination, is sufficient.

Sensitized typhoid vaccines have been advocated; they are less toxic, and it has been claimed that the resulting immunity is more effective (Gay and Claypool and others).

Plague and cholera vaccines are analogous to the typhoid vaccine and are applied in a similar fashion. The immunity following plague inoculation is generally of short duration (about three months).

Dysentery vaccine is prepared from strains of *B. dysenteriae* Flexner-Y and Shiga. Owing to the toxicity of Shiga strains it has been necessary to employ a sensitized vaccine (e.g. the British Army dysentery vaccine). *Lipovaccines* of dysentery bacilli have also been used, i.e. cultures frozen, dried *in vacuo*, and finely ground, and then incorporated in sterile olive oil; these preparations exhibit relatively little toxicity.

Pneumococcus vaccine.—In the South African mines, where pneumonia is prevalent among the native labourers, pneumococcus vaccine has proved an efficient prophylactic; a polyvalent vaccine prepared from the different serological types of pneumococcus is used (Lister).

Other infections.—Prophylactic vaccination has been also advocated for various other infections, e.g. epidemic cerebro-spinal meningitis (meningococcus vaccine), scarlet fever (polyvalent streptococcus vaccine—strains isolated from scarlet-fever cases), whooping-cough (*B. pertussis* vaccine), wound sepsis (mixed polyvalent vaccines of pyogenic cocci), influenza (mixed polyvalent vaccine of *B. influenza*, pneumococci, streptococci, etc.). The success of prophylactic vaccination must, however, depend in the first place on whether the vaccine used represents the actual causative organism or virus of the condition (but see also the non-specific action of vaccines, p. 75).

Therapeutic vaccination.—Certain chronic and localized infections are characterized by absence of a general-immunity reaction, prob-

ably owing partly to non-absorption of the bacterial products from the local lesion. In such cases vaccines prepared from the causative organism have been extensively used with varying success. Vaccination here aims at producing a general immunity reaction by injecting directly into the body the products of the particular organism (dead cultures). The injection of dead cultures may also act better as regards immunization than the products of an organism set free during the course of an infection. Typhoid vaccine, for example, may produce a much greater antibody development than the natural infection.

Both *polyvalent* and *autogenous* vaccines are employed. The former represents a number of different strains of the supposed causal organism, and such polyvalent vaccines of various organisms can be obtained commercially. The autogenous vaccine is prepared from the actual strain responsible for the lesion and isolated from the particular case.

In general, autogenous vaccines are to be preferred, but their application entails a considerable amount of detailed laboratory investigation and preparation. In some infections stock polyvalent vaccines may act as efficiently as monovalent autogenous preparations, e.g. staphylococcus aureus infections. In other cases (e.g. colon bacillus, streptococcus) individual strains are so highly specialized as regards their antigenic properties that even a stock polyvalent vaccine prepared from a large number of different strains may not represent the immunizing properties of the infecting strain. In the case of organisms which may not be readily isolated, e.g. chronic gonococcal infection, a polyvalent vaccine is frequently used. If treatment with stock vaccine fails to yield a successful result, an autogenous vaccine should, if possible, be resorted to. To use a stock vaccine, it is of course necessary that the causal organism should be known. In those cases in which a known organism is invariably associated with the condition it may be unnecessary to make a bacteriological investigation, but in many conditions various bacteria may be present, and these must be determined before initiating vaccine treatment.

In chronic furunculosis, staphylococcus aureus vaccines have proved most effective; acne (acne bacillus and staphylococci), carbuncles (staphylococcus aureus, occasionally streptococci), sycosis (staphylococcus), chronic impetigo (staphylococci, streptococci), chronic suppurating wounds, sinuses, and chronic ulcers

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(pyogenic cocci) are frequently treated successfully with polyvalent or autogenous vaccines. *In all localized chronic infections the application of vaccine-therapy constitutes a rational form of treatment*, provided that other treatment is not neglected, e.g. the surgical treatment of septic lesions. Autogenous vaccines may produce great improvement in inflammatory conditions of the urinary tract due to *B. coli*, but stock vaccines are rarely effective. Polyvalent gonococcus vaccines are frequently of considerable value in the treatment of subacute and chronic gonorrhoeal infections (urethritis, arthritis, and the vulvo-vaginitis of children). Other chronic conditions in which successful results have been recorded from therapeutic vaccination are chronic nasal and pharyngeal catarrh, atrophic rhinitis (*B. ozaena* of Perez), chronic bronchitis, asthma, whooping-cough (*B. pertussis*), chronic otitis media, chronic *M. melitensis* infections, and chronic glands (*B. mallei*). A great variety of chronic bacterial infections have been treated in this way with varying success. In chronic catarrhal conditions, e.g. rhinitis or bronchitis, where a number of different organisms are present in the muco-purulent secretions, a mixed autogenous vaccine prepared from the different organisms found in culture is employed. Chronic ringworm infections have also been treated with polyvalent vaccines obtained from different strains of the ringworm fungus. Treatment with tuberculin, which is prepared from cultures of the tubercle bacillus and consists of the products of the organisms, also represents a form of therapeutic vaccination.

The treatment of *hay fever* with extracts of pollen grains is analogous to vaccine-therapy. It has been shown that this disease is due to hypersensitiveness to the proteins of various plants, and it has been claimed that by means of pollen "vaccines" the patient can be desensitized.

Acute infections.—It is perhaps difficult to reconcile vaccine-treatment in acute conditions with the general principles of immunity, though it is possible that in some cases the injection of dead artificial cultures may effect a better immunity-reaction than the natural infection; not infrequently striking results have been obtained in this way by reliable observers, e.g. in puerperal septicæmia. Typhoid fever has been treated with vaccines by Leishman and others, and encouraging results have been reported. In acute lobar pneumonia, pneumococcus vaccines have been employed,

but with doubtful success; serologically different pneumococcus types may be associated with this condition, and vaccination is not likely to achieve results unless the vaccine is autogenous or sufficiently polyvalent. Ulcerative endocarditis, erysipelas, cellulitis, and other acute conditions have also been treated with vaccine, but this form of therapy can hardly be regarded as generally applicable to acute infections. If possible, sensitized vaccines should be employed, otherwise a specific antiserum, if available, should be administered simultaneously with the ordinary vaccine.

Dosage of vaccines.—The injections are usually given subcutaneously. Subjects vary greatly as regards susceptibility to the toxic effects of different vaccines, and vaccines vary in toxicity; the infected individual may also show distinct hypersensitiveness to the products of the specific organism, so that it is generally advisable to commence with comparatively small doses and gradually increase the amount, controlling the dosage by careful observation of the clinical condition of the patient and the reaction which follows the injections (pyrexia, malaise, etc., temporary aggravation of the existing lesion, increased discharge, etc.).

The following system of doses may generally be used: (1) 5 million organisms, (2) 10 million, (3) 20 million, (4) 50 million, (5) 100 million, given at intervals of three days provided no marked reaction occurs; then (6) 250 million, (7) 500 million, (8) 750 million, (9) 1,000 million, (10) 1,500 million, (11) 2,000 million, at intervals of seven days, and further doses of 2,000 million at intervals of ten days. If any particular dose produces a pronounced reaction, this dose should be repeated after ten days, and in no case should a larger dose be given until the lower dose in the series can be tolerated and injected without excessive reaction. Of staphylococcus vaccines, however, larger initial doses can be given, e.g. 100 million.

In acute infections it is especially necessary to use small initial doses and carefully to control the dosage. In typhoid fever, however, a first dose of 100 million, followed after three or four days by a dose of 250 million, may be given. In acute streptococcus infections the initial dose should not exceed 2 million, and in pneumonia the first injection should be 10 million.

Non-specific action of vaccines.—While vaccines generally act in virtue of their specific

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immunizing properties, instances have been recorded in which an improvement has followed their non-specific application, and it has been found also that proteid substances, e.g. albumose, proteose, milk, normal serum, injected parenterally, may favourably influence certain infections; thus, anthrax has been successfully treated by intravenous injections of normal ox-serum. The mechanism of these effects is not fully understood, though they may depend on the formation of heterogenetic antibodies as a result of the parenteral injection of alien protein (*see ante*, p. 65). They have also been attributed to hyperleucocytosis and increase of antiferment in the blood (Jobling and Petersen).

(2) PASSIVE IMMUNIZATION (SERUM-THERAPY)

Passive immunity is usually conferred by the injection of specific antisera from large animals (e.g. the horse) which have been actively immunized against the organism or its toxin. This procedure is confined to conditions definitely known to be due to a particular organism or toxin against which a powerful antibacterial or antitoxic serum can be produced in animals. Passive immunity produced in this way is temporarily but quickly developed, and is therefore applicable in the treatment of acute infections and for prophylaxis when the individual has actually been exposed or is in immediate danger of being exposed to infection, e.g. administration of tetanus antiserum in the case of wounds likely to be infected with *B. tetani* and the injection of antidiphtheria serum in the case of diphtheria contacts.

Injection may be subcutaneous, intramuscular, or intravenous, and in some cases intraspinal. *To ensure rapid diffusion of immune substances the intravenous method is essential.* With subcutaneous injection, antibodies take at least two days to attain their maximum content in the blood, and they never reach the same concentration in the blood and tissues generally as after intravenous administration. Thus, in severe acute infections a specific antiserum should be introduced by intravenous injection if possible, and injections (intravenous, intramuscular, or subcutaneous) should be repeated until improvement occurs, to maintain a high content of antibodies in the blood and tissues. When the infection is in the meninges (cerebro-spinal meningitis), or when the central nervous system is affected (tetanus), the antiserum must be injected intraspinally.

Antitoxic serums.—Those which have proved of practical value are diphtheria antitoxin, tetanus antitoxin, antivenins (snake and scorpion antivenom serums), antidyssentery serum, and "pollantin" or antipollen serum (for hay fever); the antidyssentery serum is also antibacterial.

Diphtheria antitoxin. *Prophylactic.*—Though the immunity conferred by an injection of antitoxin may last for one or two weeks only, diphtheria serum can be effectively employed for the passive immunization of contacts or of those likely to be exposed to diphtheria infection at a particular time. It is not applicable for obtaining a more prolonged immunity. Prophylactic doses (subcutaneous injection) usually given are 500 units for infants, and 1,000 units for children over one year and for adults.

Therapeutic.—The serum is usually introduced by subcutaneous injection, but it must be remembered that by this method two days elapse before the antitoxin content reaches its maximum in the blood. It is absolutely essential that the serum be injected as soon as symptoms appear, for as the disease advances larger doses are required to achieve results. It is advisable in all the more severe cases that the antitoxin be injected intravenously, or, in young children, in whom there are technical difficulties in this procedure, intramuscularly. Repeated doses are essential until the condition shows definite signs of improvement. In the milder cases an initial dose of 4,000-5,000 units is sufficient; if the local lesion is more extensive and if the larynx is involved, 10,000-12,000 units should be given, and in severe cases with dyspnoea 20,000 units should be injected intravenously. When large doses are required, especially in infants and children, the "concentrated" serums are more conveniently used and are less likely to produce toxic effects. In infants 4,000 units of the ordinary serum may be given at a single dose and repeated until the necessary amount has been administered. The question of anaphylaxis has been dealt with (p. 71).

Tetanus antitoxin. *Prophylactic.*—It is now generally recommended, particularly as the result of war experiences, that 500-1,000 U.S.A. units be injected subcutaneously as soon after the infection of the wound as possible; short of complete prophylaxis the injection tends to lengthen the incubation period. Until the wound shows definite signs of healing, the injection should be repeated at weekly intervals,

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in view of the temporary character of the immunity.

Therapeutic.—When symptoms have actually occurred and toxin has combined with nerve-cells in the central nervous system, to obtain effective results the antitoxin must be injected intraspinally and combined with intravenous or intramuscular injections. Large doses are required, and the concentrated serums are more conveniently employed. It has been recommended that doses of as much as 20,000–30,000 units should be given intraspinally (Andrewes). Park and Nicoll advocate the injection of 3,000–5,000 U.S.A. units intraspinally, and 10,000–15,000 intravenously, and the repetition of the intraspinal injections every day for three or four days.

Antibacterial serums.—These serums have been used in bacillary dysentery, meningococcal meningitis, influenza meningitis, acute lobar pneumonia, streptococcal and staphylococcal infections, gonorrhoea, typhoid fever, plague, cholera, anthrax, and tuberculosis. Certain conditions have been treated with the serums of patients convalescent from the same infection, e.g. scarlet fever, poliomyelitis, pneumonia.

Antidysentery serum (which is also antitoxic) has proved of great value in the treatment of bacillary dysentery, especially acute cases. It is essential that large doses be given (50–100 c.c.), and in severe cases that they should be given by intravenous injection. In general a "polyvalent" serum is used, i.e. an antiserum obtained by immunizing with a number of strains representing the different types of classical dysentery bacilli.¹

Antimeningococcus serum has achieved distinct success in the treatment of epidemic cerebro-spinal meningitis. It is injected intraspinally and in as large doses as possible; in an adult 30–40 c.c. of the cerebro-spinal fluid are withdrawn by lumbar puncture and an amount of serum slightly less than the quantity of fluid removed can be injected. The serum should be introduced slowly by gravity (Sophian), and during the injection the blood-pressure should be observed; a fall of 20 mm. in an adult is regarded as an indication that the maximum

amount has been injected. According to Gordon, the effect of this serum depends on its antindotoxin. In order that the bactericidal body of the serum may be effective, a certain amount of the patient's fresh blood-serum should also be injected to supply complement (Mackenzie and Martin).

Of late, *monotypical serums* (i.e. specific for the different types of meningococcus—Gordon's serological types I, II, III, and IV)—have been used in preference to the polyvalent serum, with highly successful results. The application of these monotypical serums necessitates the serological identification of the particular strain with one of the various types, and until this determination is complete the case may be treated with a polyvalent serum or with the pooled serum for types I and II, which are responsible for at least 80 per cent. of all cases.

Polyvalent pneumococcus antisera have been used in the treatment of acute pneumonia with irregular results. More recently, since pneumococci have been differentiated into several serological types, each representing different antigenic properties, a specific antiserum to type I (one of the two types most commonly found in pneumonia) has been prepared in the Rockefeller Institute and has been used with considerable success in cases ascertained by bacteriological examination to be due to this variety. Intravenous injections are recommended: 50 to 100 c.c. diluted with a half-volume of salt solution and repeated every twelve hours till 250 c.c. have been given.

Antipneumococcus serum injected intraspinally has also been recommended for pneumococcal meningitis.

Antistreptococcus serum.—This antiserum has been very largely used, but generally with disappointing results. Failure to obtain a curative effect must be attributed partly to the small doses usually injected. In acute streptococcal infections large doses should be given by intravenous injection and repeated; a small dose given subcutaneously is not likely to produce a beneficial result. It is probable that a streptococcus serum may not be sufficiently polyvalent, and the streptococcus group probably represents a large number of types all immunologically different.

Antistaphylococcus serum has also been used in acute staphylococcal infection. **Antigonococcus serum** has been advocated, especially in acute gonorrhoeal complications, and favourable results have been recorded. Intravenous

¹ The War Office Committee on Dysentery recommended the use of three curative serums: (1) a polyvalent serum prepared with Shiga's bacillus and the five types of the Flexner-Y group, to be used in the early stages before bacteriological diagnosis has been completed; (2) a monovalent Shiga serum in cases diagnosed as Shiga infections; (3) a polyvalent Flexner-Y serum in cases diagnosed as due to an organism of this group.

IMPETIGO CONTAGIOSA

injection of large doses (not less than 50 c.c.) is advisable.

The evidence in favour of **antityphoid serum** is incomplete. Beneficial effects have been recorded from the use of **antiplague** and **anticholera** serums given in large doses.

The action of **Slavov's anthrax serum** is obscure; it has no bactericidal nor antitoxic action (no toxin has been demonstrated in the case of *B. anthracis* growing in artificial culture), but its therapeutic action is undoubted. At least 50 c.c. should be administered intravenously and repeated. Cases of anthrax have been successfully treated by intravenous injection of normal ox-serum, and it has been claimed that both this effect and that of Slavov's serum are non-specific, and due to the injection of alien protein (*see p. 65*).

Antiserums to the tubercle bacilli have been prepared, but have not been extensively applied, and the results are conflicting.

Cases of **poliomyelitis** have been successfully treated by intraspinal and intravenous injection of the serum of patients convalescent from the disease (i.e. an immune serum), and cases of **pneumonia** and **scarlet fever** have been similarly treated with encouraging results.

T. J. MACKIE.

IMPERFORATE ANUS (*see* RECTUM, MALFORMATIONS OF).

IMPERFORATE HYMEN (*see* AMENORRHEA).

IMPETIGO CONTAGIOSA.—An acute superficial inflammation of the skin, beginning with small vesicles and terminating in pustules and yellow crusts.

Etiology and pathology.—Impetigo contagiosa was formerly attributed to invasion of the skin by staphylococci, but it has been shown that the initial vesicles give pure cultures of the *Streptococcus pyogenes* when cultivated in liquid media in capillary tubes, and that infection by the staphylococcus is secondary. The disease is most common in children, especially in conditions of poverty and neglect, but no age is exempt. Inoculation occurs rapidly from one part of the body to another, and from patient to patient. The primary lesion follows a slight injury or abrasion of the skin; hence the disease may result from shaving or occur in diseases accompanied by itching and excoriation of the skin, such as pediculosis, scabies, and eczema. Pediculosis capitis is a frequent cause in children. Im-

petigo also results from inoculation of folds and fissures of the skin by purulent discharges from the nose, ears, umbilical cord, or other septic focus.

Symptomatology (PLATE 14).—Although the initial lesion is a streptococcic vesicle, the streptococcus is soon outgrown by staphylococci, and the vesicles become purulent, and dry to form large, flat, brownish-yellow or greenish crusts, without much surrounding erythema, and looking as if they were stuck on the healthy skin. The clinical types vary with the part of the body affected and the age and state of health of the patient. On the scalp there are raised, greenish-black crusts matted with hair, generally accompanied by enlarged occipital glands and associated with pediculi. The face shows honey-like scabs, and the body small papulo-vesicles or pustules and crusts, often combined with symptoms of another skin disease which has become "impetiginized." Occasionally impetigo takes the form of rings (*impetigo circinata*) or bullæ (*impetigo bullosa*). In the groins, beneath the breasts, and in other parts where skin surfaces are opposed, the vesicles are easily ruptured and raw moist surfaces take the place of the characteristic vesicles and crusts. This type is called intertriginous impetigo. Similarly, behind the ears, or around the nose or mouth, chronic streptococcal fissures may be formed. "*Perlèche*" of the French is an impetigo of the angles of the mouth.

There are other special types of impetigo which differ considerably from those that have here been mentioned. The so-called *pemphigus neonatorum* is a bullous impetigo. Several infants may be infected by a midwife or nurse suffering from impetigo, or a whitlow, or septic ulcer, and the disease may thus occur in epidemic form. Small and large blebs are distributed over the trunk and limbs. The disease may end fatally owing to a specially virulent form of infection or to infection through the unhealed umbilical cord, but mild cases occur both in infants and in older children. *Dermatitis exfoliativa neonatorum*, or *Ritter's disease*, in which the whole body of the child is involved in a generalized scaly and exudative dermatitis, generally ending fatally, is probably allied to the last, and the tropical disease known as *pemphigus contagiosus* may also be included under the streptococcic infections. The term *ecthyma* has been applied to various conditions, and different varieties of microorganisms have been found in association with



PLATE 14.—IMPETIGO CONTAGIOSA.

it, but the name should be restricted to the vesicular lesions, leaving dark-brown crusts with an inflammatory areola, and sometimes followed by shallow, indolent, scar-leaving ulcers, which occur in debilitated and ill-nourished subjects as the result of invasion of the deeper layers of the skin by the streptococcus. *Dermatitis gangrenosa infantum* and the *vacciniform dermatitis of infants* may be mentioned here, but they are probably due to mixed infections, *B. pyocyaneus* and other organisms having been isolated from them, as well as streptococci. It is probable that some of the eruptions generally classed under seborrhoeic eczema, characterized by dry, red, scaly, circumscribed patches with a crinkled surface, occurring in association with purulent discharges from the ear, nose, or elsewhere, are pyogenic infections and possibly streptococcal in origin. They have been designated *chronic impetigo*. Mention must also be made of another variety of impetigo differing from contagious impetigo in that it is caused by the staphylococcus from the first, and begins as a follicular pustule and not as an epidermic vesicle. It is known as *Bockhart's impetigo*, and should be classed with boils and syphilis.

Diagnosis.—The superficial yellow, gum-like crusts without an inflammatory areola are the most characteristic lesions. When these are lifted up, a red moist surface is disclosed, which later becomes dry and glazed. There is no ulceration under the crusts as in *syphilis*, and the lesions heal without scarring. In *intertrigo* a few vesicles representing the earliest stage of the disease may be found at the margin of the moist discharging surface. *Bullous impetigo of infants* does not commonly affect the palms and soles, and the bullæ lack the coppery base of congenital syphilis. Circinate impetigo may be distinguished from *linea circinata* by the undermining of the spreading edge, the presence of superficial crusts, and the absence of mycelium in the scrapings. In *exfoliative impetigo* a certain amount of undermining of the spreading margin can sometimes be detected. The diagnosis of ecthyma and the other diseases mentioned can usually be made by a process of exclusion from *syphilitic* and *tuberculous lesions*.

Prognosis and treatment.—Impetigo of the scalp and face can generally be relieved in a few days. The crusts must first be removed by soaking them with olive oil, or bathing them with a weak solution of lysol and applying a mild mercurial ointment, such as ung. hydrarg.

ammon. 2 or 3 per cent., or ung. hydrarg. nitratis dil. 1 dr. to 1 oz. If the crusts are very extensive, boric fomentations or boric starch poultices may be required. Pediculosis of the scalp, if present, should be treated with oil of sassafras or carbolic lotion, and nasal or aural discharges or other focus of infection, such as a whitlow, treated accordingly. On the trunk similar measures are required, the strength of the application being suited to the position and amount of inflammation present. When the diagnosis has been made the treatment, as a rule, presents little difficulty. Concurrent diseases such as scabies, urticaria, etc., require the appropriate treatment. Precautions must of course be taken to prevent reinfection, and in weakly and ill-nourished children cod-liver oil and tonics should be prescribed.

S. E. DORE.

IMPOTENCE (see SEXUAL FUNCTIONS, MALE, DISTURBANCES OF).

INCONTINENCE OF URINE.—Incontinence of urine may be due to injury or local disease of the bladder or urethra, or to disturbances of their nervous mechanisms, or it may occur—particularly at night—apart from any gross disease.

1. The more common **local conditions** that allow urine to escape involuntarily are vesicovaginal fistulæ after parturition, uretero-vaginal connexions due to injury in hysterectomy and other operations, a suprapubic or perineal wound, and the impaction of a calculus that prevents the sphincter from closing. Or the incontinence may be due to local malformations, as extroversion of the bladder, epispadias or hypospadias involving the sphincter, and in females to prolapse of the bladder.

The only efficient **treatment** of incontinence due to such causes is the treatment of the primary condition.

2. Incontinence due to **injuries or diseases of the nervous system** may be primary, or it may be secondary to retention—that is, overflow incontinence and reflex incontinence. The nervous supply of the bladder and its sphincters comes from two sources: sympathetic fibres that pass from the upper lumbar segments to the inferior mesenteric ganglia, and thence by the hypogastric nerves to the bladder, excite contraction of the sphincter; while fibres of the sacral autonomic system, that take origin in the first to the third sacral segments and enter the nervi erigentes, produce, when stimulated,

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contraction of the muscles of the bladder-wall and inhibition of those of the sphincters. The sacral innervation is probably the more important in man, as it certainly is in the lower mammals. The chief afferent fibres concerned in the functions of the bladder also enter the sacral segments.

A local transverse lesion of the spinal cord above the vesical centres leads in the first place to retention of urine and distension of the bladder, as its muscular walls are for a time relaxed; if not relieved by catheterization or other means the bladder distends till the pressure within it overcomes the resistance of the sphincters, and then *overflow incontinence* results. Later, when the period of spinal shock has passed, *reflex incontinence* may develop, i.e. the bladder is emptied periodically owing to a reflex contraction of its walls and a reflex relaxation of its sphincter; but this usually occurs only if the urine remains aseptic and the general condition of the patient becomes favourable. Or there may be a *simple incontinence*, i.e. the urine may dribble away passively from a bladder that does not become over-distended, though it is rarely empty.

These symptoms may result from any local lesion of the spinal cord that produces a paraplegia; disease or injury of the vesical centre in the sacral segments, or of the sacral roots in the cauda equina, usually causes the same disturbances, but a simple dribbling incontinence is more common. Incontinence occurs in many other spinal diseases. In *tubes dorsalis* it is generally secondary to retention, but simple incontinence also occurs, or, owing to anæsthesia, urine may escape without the patient's knowledge. Not infrequently the chief complaint is that the urine dribbles away after the patient believes he has emptied his bladder. In general paralysis it may be due to associated tabetic lesions, or to the inattention and mental failure of the patient. It is less frequent in disseminated sclerosis; here the most common symptom is precipitancy or urgency of micturition—i.e. the patient cannot restrain the flow when the desire for evacuation comes, and may consequently soil his bed or clothes; but in the later stages of the disease either overflow or simple incontinence may occur. Cerebral disease rarely disturbs the functions of the bladder unless it is accompanied by coma, dementia, or some other form of mental deterioration, or unless it produces a bilateral paralysis.

In addition to that due to local lesions and

nervous diseases, incontinence may occur when the sphincter control is naturally feeble, or when the bladder is irritable. In these conditions it is usually associated with frequency of micturition (*pollakuria*). The bladder can normally contain about 200–250 c.c. of fluid at a pressure not exceeding 18–20 cm. of water, but in these states a reflex contraction occurs at a much lower pressure when the bladder is slowly filled with a fluid. The urine generally dribbles away at night, but it may escape in the day too; the difficulty in holding it is often aggravated by cold or by excitement. This form of incontinence is met with chiefly in neurasthenic and neurotic persons, many of whom have suffered with nocturnal enuresis in early life.

The **treatment** of incontinence due to nervous lesions depends in the first place on the nature of the primary disease. It is important to prevent the bladder from becoming over-distended and to avoid urinary infection. Regular catheterization may be necessary, or suprapubic drainage may be considered. Urotropine is frequently of service in helping to keep the urine clean, but the only drugs that have much influence on the sphincters are belladonna and ergot; large doses are often necessary to produce any benefit.

3. Nocturnal enuresis.—Children exhibit, as a rule, no voluntary control over the bladder in the waking state till 12–14 months old, and they generally pass water during sleep till 1½–2 years old. When older children are incontinent during sleep the condition is referred to as “nocturnal enuresis.” It may be that full control of the bladder was never attained, but frequently the history is that the incontinence developed at the age of 2½ or 3 years, after a fright, an accident, or an illness, or without any apparent cause. The urine is evacuated once or twice in the night with the force of normal micturition; it is, in fact, an involuntary micturition, not a passive dribbling away. It is often in the first half of the night that the incontinence occurs, and the trouble is usually intermittent, the bed being wet only two or three times a week. During the day there is generally full control, but frequency or urgency of micturition, indicating an irritable bladder, is not uncommon; or occasionally there may be incontinence by day.

The children may be otherwise normal, but, as Trousseau recognized, they often exhibit neuropathic taints, as excitability, timidity,

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or stammering, or there may be a retardation of mental development. Incontinence is also common in apathetic, lazy children who sleep deeply: in these it is often associated with adenoids. The urine is usually normal, but it is sometimes very acid and may contain an excess of uric-acid crystals or of oxalates. Bacilluria is rare. Nocturnal incontinence may be one of the first signs of diabetes. Threadworms should always be searched for, as they may excite the vesical reflexes that lead to evacuation. Occasionally a nervous lesion, as spina bifida, or a spastic diplegia is the cause of the trouble. Epilepsy should be considered.

The **prognosis** is good; recovery usually occurs at or before the age of puberty, but temporary recurrences after this age are not uncommon.

The **treatment** should be directed in the first place to any condition that may act as an irritant, as intestinal worms, bacilluria, glycosuria, or local abnormalities such as phimosis or urethritis. If the urine is too acid and concentrated, alkalis with potassium citrate should be given. It is very important to improve the general health of the child by tonics, exercise, and a suitable diet. A hard bed is advisable, and a bobbin may be strapped to the child's back to prevent him from sleeping on his back, as it is in this attitude that micturition is most likely to occur. Faradic or galvanic applications, with one pole over the bladder or in the urethra and the other in the perineum, are sometimes of service. Certain drugs often prove useful; especially belladonna, which should be administered in large doses for short periods—say 7-10 min. twice a day to a child of 6 or 7 years. Ergot, hyoscyanus, and strychnine are also recommended. The use of thyroid extract is occasionally very successful, especially in dull and apathetic children.

GORDON HOLMES.

INDICANURIA (see URINE, EXAMINATION OF).

INDIGESTION (see GASTRITIS, CHRONIC; STOMACH, FUNCTIONAL DISORDERS OF; DIARRHŒA; DIARRHŒAL DISORDERS OF INFANTS).

INDUSTRIAL MEDICINE.—The limits of elasticity within which health can be placed under stress and yet return to normal are considerable: hence the delicacy with which the human body reacts to its environment is seldom fully appreciated. Nevertheless, this

power of reaction is of great importance; for if stress is long maintained or frequently repeated, complete recovery may not occur. Stresses which, if of short duration, would be negligible may become influences determining those divergences from the normal, which, by converting physiological functions into pathological conditions, disturb the balance of health, originate morbidity, and result in mortality.

Increasing recognition is being given to the importance of such strains and stresses in daily life in the causation of disease. Specific microbial infection may determine the form the disease takes, but the possibility of infection occurring at all depends upon the extent to which the health, i.e. the resistance, of the host has been modified. In some cases, as when a pandemic of influenza or bubonic plague takes place, or when white races are exposed to tropical diseases, or when native races are exposed to tuberculosis, the infecting agent may be able to overcome normal resistance. But the great bulk of morbidity and mortality does not originate in this way; it depends rather on the capacity of each individual to maintain the balance of health under the environmental conditions to which he is exposed.

The capacity of the human body to maintain the balance of health under varying exposure becomes, then, a matter of the utmost importance. In the world of modern industry the exposure of those employed for at least one third of every twenty-four hours is extraordinarily diverse. The coal miner, the railway servant, the cotton operative, the cement maker, the boot manufacturer, the metal smelter, the clerk, the agriculturist, the factory mechanic, and the potter are exposed during working hours to quite different environmental conditions. The study of how the human frame reacts to these conditions is known as industrial medicine; within the room to which it holds the key lie clues to many if not most of the problems which modern medicine seeks to solve.

Elsewhere in this work description is given of diseases peculiar to occupation, such as caisson disease and miner's nystagmus. Industry cannot fail to be interested in such diseases which are peculiarly its own; they may even, as in the case of poisoning by trinitrotoluene, or tetrachlorethane, eventually throw light upon other pathological conditions, such as acute yellow atrophy of the liver; but the great value of industrial medicine is the

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opportunity it affords for searching out the reactions of the body to influences which to a less pronounced degree affect everyone.

Industrial activity and fatigue.—The physiological processes of anabolism and catabolism are represented in ordinary life by rest and activity. Both are necessary to health, but neither must be permitted to outbalance the other. Normally, activity after a certain time wanes and gives place to a feeling of healthy fatigue, i.e. the desire for rest; but the tendency, due to the struggle for existence, is for activity to be excessive, when it is followed by excessive fatigue; then, if time is not allowed for recovery, some fatigue remains to diminish subsequent activity. If this process is repeated, over-fatigue, a pathological condition, results; its presence is manifested by a tendency to succumb to sickness, the exact type of which is determined by the form of hyperactivity indulged in and by exposure to risk, whether associated with occupational environment or otherwise. Industry from its very nature affords little or no opportunity for the study of pathological results which accrue from over-indulgence in rest: but the study of industrial fatigue forms the foundation of the science of industrial medicine. Much attention has recently been given to the subject, and certain important facts have come to light.

Engagement.—Introduction to new environment, such as entering upon work at a new factory, is a considerable shock. Those least fitted to withstand it leave soon; those less fitted leave later. Rates of leaving as high as 89 per cent. in the first three months of work have been recorded; but the following recorded rates are more usual, viz. 56 per cent. in the first three months, 28 per cent. of the remainder in the second three months, 18 in the third, 13 in the fourth, 11 in the fifth, 10 in the sixth, and so on. This rate of leaving has been called the "infant mortality" of industrial life. Groups of those leaving within any period are found to have experienced during their stay higher rates of sickness than those who remain on in employment. They also experience more accidents: in one case half of the accidents at a factory occurred among the 34 per cent. of the workers with less than six months' service.

The adolescent feels the shock most; in one case boys aged 14 years were found to have stayed for a period sixteen times shorter than that of boys aged 16½ years. Young adults stay longest, after which age the rate of

leaving slowly increases with advancing years. Married women feel the strain more than single.

Environment.—Environment itself exerts great influence. There is an optimum temperature for activity, about 55° F. for active work, rising to about 65° F. for sedentary occupations, while mental activity is at its best at temperatures below 50° F. These temperatures should vary within two or three degrees about the optimum and not be monotonously fixed. The optimum temperatures are found to coincide closely with the occurrence of accidents, the incidence of which rises slowly as the temperature falls below the optimum, and rises rapidly as it rises above the optimum.

Light is an important factor in relation to health, output, and the occurrence of accidents: all of these are improved by daylight and adversely affected by artificial light; the worse the light, the more pronounced is the effect. A sound minimum standard to aim at, whether for daylight or artificial illumination, is not less than one foot-candle at the working level (in the process of weaving, the optimum is about four foot-candles at the working level), or than 0.25 foot-candle at floor level. Localized light should not shine on workers' eyes and foreheads, or headache will result; the object should be to illuminate the work, not the worker.

Atmospheric conditions as regards humidity and movement of air profoundly modify activity and health. Output falls off when the wet bulb records about 72° F.; the body-temperature commences to rise if active work is carried on at temperatures above this; risk of heat-stroke supervenes if active work is continued with a wet bulb temperature much over 80° F. or, even though no work is in progress, with a wet bulb temperature over 89° F. Rapid air movement, by promoting the evaporation of sweat, raises the above temperatures somewhat.

A healthy atmosphere should be cool rather than hot; dry rather than damp; diverse in temperature rather than uniform and monotonous; moving rather than still. These desiderata may be obtained at a temperature of 66° F. (dry bulb) with an air velocity of 8 ft. per minute, and at a temperature of 57.5° F. with an air velocity of 10 ft. per minute. Air-movement in large compartments is best ensured by the use of mechanical fans placed low down for removing air, while fresh air is admitted through inlets, three times the area of the fans, placed high up in the opposite wall.

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Adequate ventilation improves activity and defers fatigue; visible sweating should if possible be avoided. These physiological characteristics of the atmosphere can be measured by means of the katab-thermometer, an instrument devised by Dr. Leonard Hill.

Consideration is not given here to the presence in the air of workshops of injurious dusts, or harmful gases, or indeed of infectious micro-organisms, but only to the rules underlying the need for ventilation in all occupied places. Note should be taken that modern views relegate to the limbo of obscurity the idea that under normal conditions exhaled breath contains noxious constituents; thus exhaled carbon dioxide does not increase the content of that gas in the air of rooms sufficiently to affect respiration, and there are no other injurious emanations exhaled. Only when conditions exist such as were experienced in the Black Hole at Calcutta does danger arise; and then the danger is due to heat and moisture raising the wet-bulb temperature to a dangerous height.

Hours of labour.—Even though conditions of employment are satisfactory, work cannot be advantageously pursued day by day and week by week for more than a certain number of hours. Attempts to exceed this limit result in increase in lost-time due to ill-health and in decrease in output. Instances are on record of hours of work being reduced from 66·2 per week to 45·5, that is by 20·6 per cent., with an increase in output of 9 per cent.; and from 58·2 per week to 51·2, that is by 12 per cent., with an increase in output of 22 per cent. These marked instances occur only with heavy labour under the direct control of the workers, and are not to be expected for light work or work where the machine rules the pace.

Work and rest.—Investigation of the human machine at work has shown that continuous activity can only be maintained for a limited period, and that this period is shorter the more strenuous is the work. Rest-pauses must be introduced to allow for recuperation. A man who digs hard for five minutes and rests for ten will do more effective work than one who attempts to dig hard all the time. A man loading pig-iron works best if his work and rest are ordered so that he is under load less than half his time. Girls working at a press for forty minutes and resting twenty were found to have 40 per cent. greater output than when working continuously. Men working for three-quarters of an hour and resting one-quarter of

an hour, to their own astonishment did more than when they worked without any rest.

Rhythm.—Another factor of importance is rhythm in activity. Every process of life is made up of rhythm, the alternation of activity and rest; and industrial work is no exception to the rule. Rhythm, pauses and rests, gives value to the notes in the tune of life. Movements which can be made to fall in with physiological rhythm, such as that of the pulse or respiration, are performed more easily and with less fatigue. Work is, therefore, easier when the worker and not the machine sets the pace.

Healthy activity.—If the conditions of labour, hours of work, and the interpolation of rest-pauses are all well arranged, then, in addition to a fall in the rate of leaving, and a diminution in time lost due to ill-health, the following phenomena, indicative of healthy activity as measured by output, appear:—

Commencing work on Monday, output at first increases rapidly as workers warm up after the week-end rest, after which it continues to increase steadily hour by hour throughout the day; on Tuesday it starts at a higher level than it did on Monday and steadily increases hour by hour; on Wednesday again it is rather higher than on Tuesday, increasing throughout the day; and so on throughout the week. But if all is not well, output falls away on Friday, Thursday, Wednesday, or even Tuesday, as compared with the previous day; and the same thing is found to be occurring within the day, i.e. the output of the later hours is surpassed by that of earlier hours until even the first hour may be found to give the greatest result.

The position is that activity increases with practice, but at the same time activity creates fatigue which tends to slow down practice-effect. These two influences are antagonistic, and experience teaches that when fatigue gains the upper hand (as indicated by a falling off in the rate of work), a halt should be called. Otherwise fatigue will not be entirely recovered from before next day, and the output will suffer accordingly.

Records of output are valuable for measuring the results of activity; when wages are paid by piece-rates, earnings can be used for this purpose. A steady falling off of earnings on the part of any worker should always be noted as a danger signal of impending breakdown.

Lost-time.—The reactions of the human organism can in these ways be closely watched

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and studied. When the limits of healthy elasticity are overstepped, at first a stage of disinclination for work appears, and workers, for no particular reason, leave or absent themselves. They are still within the realm of preventive medicine: they have nothing definite the matter, and frequently earn a reputation for being work-shy or lazy; but in fact they have lost the urge forward to catabolic activity and the pleasure associated with exercise which nature has made an integral part of perfect health. Irregularity in attendance is the first indication that health is feeling the strain; the amount of time so lost varies considerably in different occupations; a loss of 10 per cent. of possible time is not unusual in some factories, even though 5 or even 4 per cent. may be the rule in other factories similarly employed. The distribution of hours of labour exerts an important influence; a factory working a before-breakfast spell always experiences more lost-time than one, working the same number of hours per week, which starts after breakfast.

The irregularity of work occasioned by lost-time causes industrial establishments to pay considerable attention to the matter; and records are kept which distinguish between absenteeism due to so-called "avoidable" causes and that due to unavoidable causes such as certificated sickness. Careful inquiry has shown that these two forms of absenteeism wax and wane together, from which the inference may be drawn that both depend upon the same factor—ill-being. Hence records of lost-time in industry may be taken as a direct indication of the strain to which health is being subjected.

Industrial morbidity.—Investigations into the prevalence of sickness among those industrially employed establish that sickness is a function of age. Males under 15 years of age suffer as much as men aged 25–35; but males between 15 and 25 years of age suffer less than either juveniles or older men. After 35 years of age sickness increases with age until males over 65 years of age suffer twice as much as those between 25 and 35. In the case of females, sickness progresses steadily with age from 15 years onwards. Under 20 years of age females suffer less than males, but from 20 to 50, i.e. during the child-bearing period, they suffer more than males. After 50 years of age they again suffer less than males. These facts are important in relation to the industrial employment of women.

Information as to the kinds of sickness which occur is less precise. They certainly vary with occupation. The chief causes of sickness are various forms of rheumatism and local pains, respiratory affections, and injuries. These three form more than half the total. Rheumatism and respiratory troubles are particularly associated with exposure to climatic conditions and heavy physical exertion; but strenuous work before hot furnaces, unless care is taken to remove sweat-laden clothes, is a cause of rheumatism. No relation has been established (contrary to the popular idea) between the prevalence of respiratory diseases and any tendency to succumb to pulmonary tuberculosis; phthisis and respiratory diseases are only found associated together in connexion with exposure to the inhalation of silica dust, but here the exposure is the underlying cause of both troubles.

Much yet remains to be done to determine the forms of ordinary sickness prevalent in different occupations in order to ascertain the influences which place strains upon the various systems of the body, and the way in which mortality results from these strains. Exposure to risks peculiar to occupation, such as risks of anthrax from manipulating wool, hides and skins, of plumbism in the lead industries, of carbon-monoxide poisoning in coal mines, from nitrous fumes in making explosives, of mercurialism from exposure to mercury, and of certain forms of industrial dermatitis, are only of minor importance compared with this wide aspect of industrial medicine.

Notification of industrial disease.—Nevertheless, notification of cases of industrial poisoning has proved of great value. The diseases which must now be notified under the Factory and Workshops Act to the chief inspector of factories, by every medical practitioner, are anthrax, or toxic jaundice, contracted in a factory or workshop, and poisoning by lead, phosphorus, arsenic, or mercury. The best instance of administrative action resulting from the information so obtained is that of lead poisoning. Notification enabled the prevalence of cases to be distributed according to occupation and to the number of persons at risk. Then the prevalent influence in causation—the inhalation of dust or of fumes containing soluble lead compounds—became manifest. Action taken to protect workers against this risk, either by substituting wet methods for dry, by removing dust at the point of generation by means of localized

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exhaust draught, or by protecting workers with respirators, reduced the prevalence of notified cases in the United Kingdom from 1,058 cases in 1900 to 144 in 1918, even though during the intervening period the numbers employed in the industries concerned had considerably increased. Other influences, such as close medical supervision, improved personal hygiene, and better feeding have helped; but without doubt protection against risk has been the ruling factor.

Compensation for industrial disease.—

The inclusion in the schedule to the Workmen's Compensation Act, 1906, of certain occupational diseases so as to entitle sufferers to compensation has provided another source of information; in some cases, as in poisoning by tetrachlorethane, preventive measures have been found effective. This volatile fluid, being a solvent of cellulose, was used as a constituent of "dope" applied to aeroplane wings. Workers inhaling the vapour contracted jaundice and in some cases died from acute yellow atrophy of the liver. Prohibition of the use of tetrachlorethane abolished the disease.

The inclusion of dermatitis and ulceration of the skin, if of industrial origin, in the schedule has caused attention to be paid to these conditions with advantage, by defining the causation, in certain instances, and so pointing to the means for prevention. Thus, substances which dissolve the fats from the skin, such as benzene, turpentine, and certain oils used in engineering shops and in the manufacture of roll tobacco, leave the skin dry after long exposure; it then cracks, when microbes invade the underlying tissue and set up a dermatitis which varies in form with the invading organism. Personal cleanliness and use on the skin of lanolin and castor oil in order to restore the removed fat prove effective preventives. Hygroscopic substances, such as common salt, potassium bichromate, and some arsenic compounds form another group causing dermatitis; these substances, if they come in continuous contact, through skin cracks or abrasions, with the underlying tissues, cause local necrosis by abstracting fluid. Indolent, painful ulcers of the skin result, or perforation of the nasal septum if the substances are inhaled as dust. Prevention here takes the form of soaking the exposed parts, usually the hands, frequently in warm water to remove the hygroscopic substance, or of wearing respirators to guard against inhalation.

The list of diseases scheduled is as follows:—

Anthrax.

Mercury poisoning or its sequelæ.

Phosphorus poisoning or its sequelæ.

Arsenic poisoning or its sequelæ.

Lead poisoning or its sequelæ.

Poisoning by benzene and its homologues, or its sequelæ.

Poisoning by nitro and amido derivatives of benzene and its homologues (trinitrotoluene, aniline, and others), or the sequelæ.

Poisoning by dinitrophenol or its sequelæ.

Poisoning by nitrous fumes or its sequelæ.

Dope poisoning; that is, poisoning by any substance used as or in conjunction with a solvent for acetate of cellulose, or its sequelæ.

Poisoning by tetrachlorethane or its sequelæ.

Poisoning by carbon bisulphide or its sequelæ.

Poisoning by nickel carbonyl or its sequelæ.

Poisoning by *Gonioma kamassi* (African boxwood) or its sequelæ.

Dermatitis produced by dust or liquids.

Ulceration of the skin produced by dust or liquids.

Ulceration of the mucous membrane of the nose or mouth produced by dust. Chrome ulceration or its sequelæ.

Epitheliomatous cancer or ulceration of the skin due to tar, pitch, bitumen, mineral oil or paraffin, or any compound, product, or residue of any of these substances.

Ulceration of the corneal surface of the eye due to tar, pitch, bitumen, mineral oil or paraffin, or any compound, product, or residue of any of these substances.

Scrofula epithelioma (chimney-sweep's cancer).

Compressed-air illness or its sequelæ.

Cataract in glassworkers.

The disease known as miner's nystagmus, whether occurring in miners or others, and whether the symptom of oscillation of the eyeballs be present or not.

Subcutaneous cellulitis of the hand (beat-hand).

Subcutaneous cellulitis over the patella (miner's beat-knee).

Acute bursitis over the elbow (miner's beat-elbow).

Inflammation of the synovial lining of the wrist-joint and tendon-sheaths.

Ankylostomiasis.

Glanders.

Telegraphist's cramp.

Writer's cramp.

Accidental gassing.—Another group of diseases to risk of which exposure exists in industry is that due to various toxic gases such as carbon monoxide contained in water gas and blast-furnace gases, and formed when

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explosions occur in mines; nitrous fumes generated in making high explosives and in etching copper plates; arseniuretted hydrogen, formed when metals are pickled with impure sulphuric acid; phosphoretted hydrogen which may arise from ferro-silicon; sulphuretted hydrogen, generated in chemical works; and bisulphide of carbon used in rubber works. The diseases which are caused by these gases do not necessarily appear in the Schedule given above because, since the moment of exposure can be defined, they are considered as accidents within the meaning of the Act, and all accidents are already included for compensation.

The effect produced varies with the gas: thus, carbon monoxide causes unconsciousness immediately; while nitrous fumes irritate somewhat but hardly enough to give warning that a few hours later profuse mucus-secretion will take place in the air-passages, drowning the patient some thirty-six hours after exposure. Arseniuretted hydrogen, again, may hardly betray its presence at the time, but sickness may supervene in a few hours, to be followed by serious or even fatal results from jaundice and liver destruction a fortnight later.

Occupational mortality.—The most reliable information available to-day for throwing light upon the effect of environment upon health lies in statistics of occupational mortality. These are based upon information with regard to occupation obtained at each census, against which are placed deaths occurring in each occupation during three years—the year before, the year of, and the year after the census. The statistics indicate that the mortality experienced in different occupations is extraordinarily diverse; for example, agricultural labourers have a comparative mortality-rate of 470, Nottingham coal-miners of 570, limestone masons of 753, printers of 773, tailors of 799, cotton operatives of 811, shoemakers of 820, iron and steel manufacturers of 837, fishermen of 858, Lancashire coal-miners of 941, edged-tool makers of 1,010, sandstone masons of 1,427, costermongers of 1,507, tin-miners of 1,579, and general labourers of 2,301. Examination of the causes of death which go to make up these rates discloses that in different occupations different diseases are especially prevalent; thus the Lancashire coal-miner suffers in excess from pneumonia and accidents; the printer, the tailor, and the shoemaker suffer in excess from phthisis; while the edged-tool

maker, the sandstone mason, and the tin-miner suffer in excess from tuberculous silicosis and other respiratory diseases.

Full use has not yet been made of these data, but one instance may be chosen to indicate the method and the value of extended investigation based on mortality figures.

Phthisis in occupation.—During juvenile life phthisis is rather more prevalent among females than among males. On the other hand, during adult life, when males are industrially employed, it is more prevalent among males, and becomes more and more so with advancing years; this change-over occurs later in agricultural districts, and is never excessive. There are two possible causes for this: (i) industrial work, by placing a strain upon health, may lower the resistance to infection; (ii) aggregation of individuals in factory life may increase the possibilities of infection. One or the other may be in the ascendant; or both may act together.

An instance of strain lowering resistance is to be found among the *pneumonoconioses*. Here, workers exposed to the inhalation of one particular form of dust, silica dust, experience a high mortality, not only from respiratory diseases, such as pneumonia and bronchitis (in common with others exposed to the inhalation of dusts insoluble in the fluids of the body), but also from fibroid phthisis. The strain in this case is associated with a peculiar action on the part of silica, which sets up a condition known as pulmonary silicosis; persons so affected are particularly liable to succumb to pulmonary tuberculosis. Some among the many industries in which this disease is to be found are tin-mining, gold-mining, dressing sandstone, the refractories industry (due to exposure to ganister dust), making pottery (due to flint dust), grinding articles on sandstone wheels, dressing granite, lead-mining (when the ore lies in a quartz mother-rock), making and using millstones, flint-knapping; all of which expose the workers to the dust of silica. The reason for this is probably that silica is converted in the body into silicic acid which reacts with lung tissue to form silicotic fibrosis; it also acts on the kidneys while being excreted and sets up nephritis; hence groups of workers who suffer from tuberculous silicosis are also found with a high mortality from Bright's disease.

The clinical condition associated with silicosis is dealt with elsewhere (see *PNEUMONOCONIOSES*); here attention need only be drawn

to the facts that it usually terminates in pulmonary tuberculosis, that the resulting death-rate occurs at a later period of life than that for ordinary phthisis, and that it is found associated with high death-rates from other respiratory diseases, such as bronchitis and pneumonia, and from Bright's disease.

The high mortality from phthisis among printers, shoemakers and tailors is, on the other hand, an instance of the aggregation of individuals increasing the possibilities of infection. The high mortality from phthisis in this case is not necessarily associated with high mortalities from other respiratory diseases or from Bright's disease. Other influences, such as working in an atmosphere which lacks bracing qualities, may contribute to the result; but since the phthisis rate in the industries named is higher than in others, for example cotton weaving, where the atmospheric conditions are quite as adverse, the paramount influence must be ascribed to increased possibilities of infection.

A clear understanding of these two causes of phthisis indicates the means for prevention. In the first case, inhalation of silica dust must be reduced to a minimum, either by altering processes, so that dust is not generated (the wet grinding of flint is an instance in point), or by removing dust, as it is formed, from the atmosphere of workplaces (accurately designed localized exhaust draught is the means for the purpose), or by supplying the workers with air free from dust (as in the process of sand-blasting castings), or, as a last resort, by wearing respirators capable of filtering out the dust (but no absolutely satisfactory respirator has yet been designed). In the second case, the need is to avoid crowding as far as possible, to arrange that operatives do not stand or sit close together or facing one another, and to ensure air-movement by the introduction of a sufficient supply of fresh air.

Consideration in neither of the cases quoted is given to the elimination of infectious cases from industrial environment. Action on these lines belongs rather to the tuberculosis service than to industrial medicine. But when factory medical officers are the rule rather than the exception, they will need to give careful consideration to the question.

Conclusion.—Probably there is no form of ill-health and disease affecting adult life which is not influenced by occupation. Certainly occupations provide wide variations in com-

parative mortality-rates from different causes of death, such as the following:—

Diseases of the nervous system: file-maker, 194; gamekeeper, 34. *Diseases of the circulatory system:* general labourer, 294; gardener, 82. *Cancer:* chimney-sweep, 136; tanner, 33. *Phthisis:* tin miner, 838; railway engine-driver, 63. *Pneumonia:* copper manufacturer, 206; silk manufacturer, 25. *Bright's disease:* lead manufacturer, 149; ironstone miner, 8. *Accidents:* coal miner, 123; bookbinder, 13.

Investigations on the lines indicated above into such variations, together with field inquiries into the environment and conditions of work with which they are associated, cannot fail to disclose problems capable of being put to the test of laboratory experiment. Cancer in particular offers a favourable field; but the distribution of the disease according to the part affected is the first need; already there is evidence that epithelium may be sensitized to the disease by exposure to some product or products of vegetable distillation. Of this examples are to be found in soot, which causes in chimney-sweeps scrotal cancer; in pitch, which originates epitheliomatous warts among briquette makers; in crude paraffin, which causes shale-oil workers to suffer from paraffin epithelioma; in aniline compounds, which set up in dye-workers malignant papillomata of the bladder; and in tobacco smoke, which nearly certainly is the origin among males of their high death-rate from cancer of the lip and buccal cavity.

An attempt has been made, not to cover the whole field of industrial medicine, but just to sketch the importance of the subject, and to show how the varied exposure of industrial life may reveal influences which, by modifying metabolic activity in this way or in that, may overstrain the elasticity of health, and so pave the way to ill-health and disease.

EDGAR L. COLLIS.

INFANT FEEDING.—This subject may be considered under the following headings:—

1. Natural feeding.
2. Mixed feeding.
3. Artificial feeding.
4. Wet-nursing.
5. Premature and weakly infants.
6. Weaning and feeding after infancy.

1. **Natural feeding.**—It is of the greatest advantage both to mother and offspring that the infant should be suckled, not hand-fed. Fortunately, most women are alive to this, and are

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anxious to nurse their infants if possible. There is no certain means of ensuring that an expectant mother will be able to suckle her child, nor is it possible by examination of the breasts or otherwise to foretell the event. The most that can be done is to attend to her general health and to the condition of the breasts and nipples, and to assure her that in all probability lactation will go on normally. So far as the general health is concerned, all that is necessary is that the ordinary rules of hygiene as to food, fresh air, and exercise be attended to. After delivery, a liberal diet should be given, including plenty of fluid such as gruel and milk. During the normal puerperium the appetite is, as a rule, excellent, and there is no reason for restricting the diet. The infant should be put to the breast within twelve hours of delivery, because the act of suckling is the natural stimulus to the function of the mamma. Thus a vigorous infant has a better chance of being nursed than a feeble baby which is disinclined to suck. The secretion of milk is not, of course, properly established for several days, perhaps a week, after delivery, and when it is long delayed the question of giving the child some additional nourishment, or of feeding by hand, will arise. Here the decision depends largely on the size and strength of the child, and it is impossible to lay down any hard-and-fast rules.

Certain points should, however, be noted : (1) During the first few days, if the infant is getting very little from the breast and is restless and fretful, he requires fluid rather than food, and only water sweetened with a little sugar or saccharin is needed. (2) Until artificial feeding is definitely decided upon, the infant should be fed from a spoon, not a bottle, because the effort required to suck from a bottle is so much less than that required to suck from the breast that the infant is apt to refuse the latter. (3) When once bottle-feeding is begun because the breast-milk is scanty, the latter will probably fail altogether on account of the lessened stimulus to secretion. (4) It is of very decided benefit if the baby can be suckled during the colostrum period. At this epoch every day is a distinct gain.

No drug can be relied upon to promote the flow of milk. One which may be tried is thyroid extract ; one 5-gr. tablet may be given, and, if no ill effects follow, the dose should be increased to two or three in the day.

During the month or two preceding delivery the nipples require attention. If they are small

and flattened they should be manipulated daily so as to improve their shape, and pressure from corsets, etc., should be avoided. They should be carefully cleansed daily to remove sebaceous material, but attempts to harden the skin by bathing with spirit are of doubtful value. From the very beginning of lactation great care should be taken to prevent cracking. Cracks or fissures are extremely painful and, next to insufficiency of milk, are the most common cause of inability to nurse. Scrupulous cleansing and drying of the nipples after each nursing is the best prevention. When a crack has developed, one of the simplest methods of treating it is to use the old-fashioned lead nipple shield. This consists of a flanged thimble of thin lead, which is worn in the intervals of suckling. The nipples must be carefully cleansed, and the shield should be boiled once a day. The action of this shield is twofold : it is a splint, giving the cracks complete rest and preventing the nipple from being pressed on or rubbed by the clothing ; it also keeps the cracks moist and prevents crusts from forming. The improvement which results, even if the nipple is badly fissured, after one of these shields has been worn for a couple of days, and the comfort which it gives, are astonishing. It is much better than the ordinary nipple shield, which is used mainly as a protection during suckling.

Regularity in nursing is of prime importance. During the first two days after birth the child should be given the breast every six hours ; during the third day, every four hours ; and thereafter, when the flow of milk is established, at more frequent intervals. The infant should be nursed at each breast alternately, and the duration of the feed ought not to exceed twenty minutes. The following *schedule of times* is that commonly in vogue :—

<i>Age</i>	<i>Feeds</i>
1-6 weeks . .	Ten 2-hourly feeds from 6 A.M. to 12 P.M.
6-10 weeks . .	Eight 2½-hourly feeds from 6.30 A.M. to 12 P.M.
10 weeks and later	Six 3-hourly feeds from 7 A.M. to 10 P.M.

The important point to insist upon is the long interval at night. It is nearly always possible to train an infant to go without food for a period of six hours, if training is begun early. The above schedule, or a modification of it, represents the ordinary custom in this country, but in many cases the 2-hourly

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feedings can be dispensed with, and a beginning made during the first week with 3-hourly or 4-hourly feedings. If the prejudice in favour of frequent nursing can be overcome, this plan ought to be adopted. The infant gets the breast only four or five times in the twenty-four hours, at intervals of three or four hours.

While the mother has enough milk there is seldom any difficulty in rearing an infant, and its progress is usually so evident that regular weighing is not absolutely necessary. This, however, is a useful guide, as cessation in gain in weight is often the first sign that the milk is becoming insufficient. It is therefore advisable to weigh the infant once a week, or once a fortnight.

Provided that progress is maintained, the infant ought to have nothing except breast-milk until it is seven months old, and in many cases it need not be weaned until the ninth month. As a rule, however, it is well to replace one or two nursings by substitute feeds from the seventh month. When hand-feeding is begun at this age the child should be taught to drink from a spoon or cup, and no feeding-bottle should be used. It is best to begin with boiled milk diluted with half its bulk of water, and, after this has been used for a fortnight or so, to give some starchy food such as gruel in addition, as is described in connexion with artificial feeding. More and more breast-feeds are replaced as time goes on, and the child should be weaned completely in about six weeks. In many cases, however, one or two nursings are continued for a considerable time after hand-feeding has begun.

In spite of a good deal that has been written, it must be confessed that when an infant suffers from indigestion or does not thrive at the breast, very little can be done to render the mother's milk more suitable. The first thing is to ascertain whether she is really careful in giving the breast regularly. Irregular or too frequent nursing is the commonest cause of difficulty, and gives rise to the ordinary symptoms of indigestion—colic, vomiting, and diarrhoea. If the milk is deficient, either in quantity or in quality, the child ceases to gain, is dissatisfied when the breast is empty, and displays signs of hunger. On the other hand, when the milk is too rich in fat, colic and vomiting, or even fat-diarrhoea, may result. An effort may be made to improve deficient milk by increasing the protein in the mother's diet and by making her rest. If there is reason

to think that the milk is too rich, the proteins in the diet should be restricted, the fluids increased, and more exercise enjoined. If there is no improvement after a week or a fortnight, it is better not to delay substitute-feeding. Neither malt extract nor malt liquors seem to have much effect in promoting the flow of milk.

Direct examination of breast-milk is of little practical use on account of the great variations which occur within normal limits. The quantity secreted can be estimated by weighing the infant immediately before and immediately after a nursing, and may afford some guidance in cases where progress is not satisfactory. The following figures, based on five carefully observed cases, are quoted from Holt, and may be taken as representing the normal range:—

Age	Quantity at one nursing
First week	1-1½ oz.
Second week	1-3 "
Fourth week	1½-4½ "
Eighth to eleventh weeks	2½-5½ "
Fourth month	3-6 "
Sixth month	4-7 "

Of the **digestive disturbances** in breast-fed infants, *vomiting* is the most important from the practical standpoint, because it is not infrequently due to congenital hypertrophy of the pylorus. Vomiting during the first month in a breast-fed infant (especially if the child is a boy) ought always to arouse the suspicion of this, and to treat such a case as one of "the mother's milk not agreeing" is a serious mistake, on which the infant's chance of life may hinge. Apart from this, vomiting is often due simply to overfilling the stomach, in which case the overflow is regurgitated without effort; the remedy is obvious. Dyspeptic vomiting is mostly due to irregular or too frequent feeding, or, in some cases, to the milk being too rich.

Colic may afflict breast-fed infants. A small dose of castor oil will usually give relief. In many cases chilling of the body is the cause.

Constipation is not rare. A distinction should be made between infrequent passage of normal motions and the passage of hard, lumpy stools. If the motions appear fairly normal and their passage is unattended by discomfort, aperients are not needed, even if the bowels act only every third day. In such a case the milk supply is probably inadequate and

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the child not thriving; or the condition may be due to habit. If the motions are hard and dry, an aperient will probably be required. Fluid magnesia, olive oil, and malt extract are suitable. Castor oil and calomel ought not to be used as habitual purgatives.

It is a popular belief that many drugs, and certain foodstuffs, especially such as contain aromatic substances, when taken by a nursing woman, may affect the milk and cause symptoms in the infant. The only drugs which require to be considered in practice are salicylates, iodides, rhubarb, senna, and thyroid extract.

The reappearance of the menses during lactation does not, as a rule, necessitate weaning unless the mother's health suffers or the milk begins to fail. The milk secreted during a menstrual period seldom disagrees.

Lactation is contraindicated by most organic diseases of the mother, particularly tuberculosis and albuminuria. The supervention of pregnancy is also a reason for removing the child from the breast. On the other hand, temporary acute illness, such as tonsillitis, does not necessarily interfere with suckling.

2. Mixed feeding.—When a woman has some milk, but not enough for the infant's needs, the child should be fed partly by hand and partly at the breast. This is certainly better than artificial feeding. The substitute-feeds should be prepared as described in the next section, and should be given alternately with breast-feeds, or otherwise, as is found convenient.

3. Artificial feeding.—While it is easy to rear a baby at the breast without any knowledge of the composition and peculiarities of human milk, it is difficult to manage hand-feeding satisfactorily without some definite idea of the foods we are using. Human milk is the standard on which artificial foods are, as far as possible, modelled. At the outset, however, we are met by the fact that the milk of every animal varies within wide limits—not only at different periods of lactation, and from one nursing to another, but even during the progress of each act of suckling. Statements of the "average" composition of milk, such as are given below, are therefore merely arithmetical means, and should be interpreted only as such. It follows that all modifications of cow's milk only approximate to the standard, and, when we consider the uncertain composition of the material we use, it is apparent that undue refinement in calculations of percentages

are often misleading. The real advantage of giving detailed instructions as to the exact composition of an infant's dietary is that only in this way can we intelligently alter it to suit the child—not, as is sometimes thought, that a complicated mixture, by more closely resembling human milk, has any intrinsic superiority over a simple one.

We have therefore to consider—

- (1) The composition and characters of human milk
- (2) The composition and characters of cow's milk.
- (3) The amount of food an infant needs.
- (4) The feeding of a healthy baby.

Although digestive and nutritive disorders do not fall within the scope of this article, it will be necessary to refer—

- (5) To some difficulties which may be met with in artificial feeding; and
- (6) To modifications of milk, etc., which are useful in special circumstances.

(1) Composition and characters of human milk.—The following table (Holt) is based on recent analyses:—

<i>Common, healthy variations</i>				<i>Average</i>
Protein	. 1	to 2.25	per cent.	1.25
Fat	. 3	" 5	"	3.5
Sugar	. 6	" 7	"	6.5
Salts.	. 0.18	" 0.25	"	0.2

The *protein* consists of casein and soluble albumin in the proportion of 4:5. The *fat* exists in the form of minute globules of neutral fats, in which olein predominates, fatty acids being scanty. The *sugar* (lactose) and the *salts* are in solution. The composition of milk alters during lactation. During the first few days it is called colostrum, and differs greatly from that secreted later. Colostrum is deep yellow, from the presence of fatty degenerated cells (colostrum corpuscles); its protein is high and its fat and sugar are low. During the rest of lactation, individual differences are so great that only a very general statement can be made: that, on the whole, the percentage of protein tends to fall, and the percentage of sugar to rise; the percentage of fat rises or falls quite irregularly.

Human milk is alkaline to litmus; it contains ferments, the function of which is unknown; and it is sterile. During the colostrum period it contains antigens, which are lacking in the body fluids of the new-born infant. Hence, probably, the supreme importance of lactation during the earliest days of life.

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(2) **Composition and characters of cow's milk.**—Cow's milk of good quality has the following composition:—

	Percentage
Protein	3·5
Fat	3·5-4
Sugar	4·5
Salts	0·75

The *protein* consists of casein and soluble albumin in the ratio of about 7 : 2, and the *fat* contains more of the lower volatile fatty acids than are found in human milk. The *lactose* differs only in amount. The *mineral salts* are much more abundant—a point on which considerable stress is now laid. The reaction to litmus is amphoteric. As it reaches the consumer, cow's milk swarms with bacteria.

We know only too well that cow's milk is far less suited to the infant than human milk, but we do not know why this is the case. Hitherto it has been customary to ascribe the difficulty to the most obvious contrast with human milk, viz. the large amount of casein, and for years efforts to modify this peculiarity dominated the whole theory and practice of infant-feeding. It is, however, very doubtful whether the alleged indigestibility of casein is a reality—certainly it is not nearly so important a factor as is popularly supposed. It is impossible in this article to discuss the subject fully, but the modern hypothesis, which is largely due to Finkelstein and his school, may be stated shortly thus: Neither protein, fat, nor sugar is *per se* responsible, but, owing to its high mineral content, the whey in which they are presented to the digestive cells is an unsuitable medium for the proper functioning of these cells. If, in consequence, the cells are overtaxed, either because they are innately feeble, or because too much food is offered to them, their function breaks down as regards fat, or sugar, or both. Failure to deal with fat leads to malnutrition; failure to deal with sugar, to dyspepsia.

The question of the bacterial contamination of milk is more conveniently referred to later.

(3) **Amount of food an infant requires.**—Our knowledge of the amount of food a baby requires is drawn from two main sources—(a) direct observations of the quantities of milk consumed by infants showing normal weight-curves, and (b) the method of calorimetry. An infant requires on an average one-seventh of its body-weight of cow's milk daily; some infants need as much as one-fifth, while others thrive well on as little as one-tenth. The first figure

gives a basis for trial: thus, a 7-lb. baby ought to receive about 16 oz. of milk daily.

The employment of the caloric method as a means of stating quantities has recently become fashionable; it may appear complex, but it is really very simple. The daily requirements of a baby from birth to the sixth month are about 100 Calories per kilo of body-weight (45 Calories per lb.); during the second six months, about 80 Calories per kilo (36 per lb.). The caloric value of any food being known, the amount needed can be easily deduced. The caloric value of a food is calculated from its percentage composition by multiplying the protein and carbohydrate by 4·1, and the fat by 9·3. The sum of the products gives the Calories per 100 grammes (3½ oz.). One litre of cow's milk containing 4 per cent. of fat yields some 700 Calories, enough for a baby weighing 7 kilos, or 15 lb. Adopting the simple rule of one-seventh of the body-weight, we get the same result: an infant weighing 15 lb., or 240 oz., will require about 35 oz. of milk. Thus, all that the caloric method does is to give us a means of statement by which all foods are reduced to a common standard and can be compared with one another. So long as we use only cow's milk it is unnecessary to state quantities as Calories, but when a complicated modification of milk or other food is employed it is desirable to have some idea of its caloric value, otherwise it is difficult to regulate the quantity with the assurance that it is neither too large nor too small.

The size of each feed depends to some extent on the size of the stomach. An easily remembered rule is to allow one ounce for each month up to the seventh or eighth. More than this can easily be given without overfilling the stomach, because before a meal has ended some of the food has already passed into the duodenum.

(4) **Feeding of a healthy baby.**—Cow's milk of good quality should be used, and only when it fails ought other milk preparations to be tried. Provided that the infant's digestion has not been upset at the very outset, and that a certain amount of common sense is employed, there is very seldom any difficulty in rearing a baby by hand on cow's milk.

The milk ought to be sterilized by boiling. The term "sterilization of milk" is very loosely used, and does not, as a rule, imply sterilization in the bacteriological sense, but only that the milk has been heated enough to destroy the ordinary milk bacteria and

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pathogenic organisms, particularly the tubercle bacillus. Sterilization by the addition of antiseptics, as hydrogen peroxide, is inferior to sterilization by heat, and milk treated antiseptically is unfit for infants. While it may be conceded that the bacterial contamination of milk is of less moment so far as the diarrhoeal diseases of infancy are concerned than was at one time believed, recent work conclusively shows that tuberculosis is frequently conveyed by milk. No doubt the prevalence of milk infections varies greatly in different localities, and possibly they are more common in Scotland than in England, but, when we consider that, on an average throughout the country, at least one sample of commercial milk out of every ten contains tubercle bacilli; that in some localities the proportion is much higher; that much of the tuberculosis of infants and young children is due to the bovine bacillus; and that tuberculosis during the first year of life is almost invariably a rapidly fatal disease, the conclusion is unavoidable that raw milk is a dangerous food.

The objections to heating milk are negligible in comparison; they fall under two (or three) heads. First, it is said that boiled milk is less nutritious than raw milk; but, so far as direct evidence goes, Dr. Lane Claydon's work supports clinical experience that this is not the case. Second, it is urged that raw milk is a more "natural" food, as it contains ferments which heating destroys; it is even alleged that minimal doses of tubercle bacilli may not be deleterious but beneficial by evoking an acquired immunity. Conjectures such as these, conceivably true, but lacking scientific demonstration, do not outweigh the proved dangers of raw milk. Third, there is the undeniable fact that sterilized milk is one of the foods which may produce scorbutus. This disease, however, is not only rare, but easily prevented and cured, and the remote possibility of its occurrence does not counterbalance the security gained by boiling.

Milk, therefore, should be boiled for from five to ten minutes; it should be chilled rapidly in cold water, and kept covered in a cool place. It is in many ways more convenient to use a sterilizer and prepare at one time the whole day's feeds in separate bottles. The original Soxhlet pattern is as good as any other. Pasteurization (exposure to a temperature of 156° F. for twenty minutes) is sometimes recommended as better than boiling, but it is rather uncertain and has no real advantages.

Boiling produces certain changes in milk besides killing bacteria. The ferments are destroyed and the antiscorbutic vitamin is diminished; some of the lime salts are precipitated, and in consequence a less dense curd forms on the addition of rennin; the soluble albumin is coagulated, and the taste is altered. These changes are to some extent obviated by boiling in a closed vessel and chilling quickly; the alteration in taste is then less, and the soluble albumin does not form a skin on the surface but remains suspended in the whey. As routine methods of infant-feeding, two may be advised—(a) milk diluted with water, the mixture being gradually increased in strength; and (b) whole milk (Budin's method).

(a) *Feeding with diluted milk.*—The object of diluting milk is to diminish the amount of casein and to assimilate its composition to that of mother's milk. For purposes of calculation woman's milk is taken as containing 1.5 to 2 per cent. of protein, 4 per cent. of fat, and 6 per cent. of sugar; cow's milk, as containing 4 per cent. of each constituent. In most cases water is the best diluent; barley-water is apt to cause fermentation and upset the digestion; and since we do not, nowadays, regard the casein curd as the essential cause of difficulty, the supposed mechanical effect of barley-water may be disregarded.

To begin with, we order 1–1½ oz. of milk with an equal bulk of water every three hours. This mixture is deficient in fat by 2 per cent., and in sugar by 4 per cent.; it is not necessary to correct the fat, but the sugar should be raised to 5 or 6 per cent. by the addition of 1½ level tablespoonfuls of milk or cane sugar to every 20 oz. of the mixture (one day's feeds). Milk sugar is probably preferable to cane sugar, but there is not much to choose between them, and where expense is a consideration cane sugar may be used. In the majority of cases this mixture will suit perfectly well: the points which require attention are (i) the character of the motions; (ii) whether there is any colic or vomiting; (iii) whether the baby is satisfied; and (iv) the weight from week to week. In all forms of hand-feeding, regular weekly weighing is of the greatest assistance; a gain of 4 or 6 oz. per week should be aimed at. A weekly increase much greater than this is not desirable; it suggests that the child is being overfed, which is one of the chief dangers in using cow's milk.

As a rule, it will be necessary to increase the food about the third or fourth week; this

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may be done either by giving more of the above mixture or increasing its strength. On the whole, it is preferable to adopt the latter course, the object being to get the infant on to a fairly strong milk mixture, or even whole milk, comparatively early. By this plan the need for giving cream is done away with, and the feeds are less bulky. We may, therefore, increase the strength to two parts of milk to one of water, adding 3 per cent. of sugar. It is often as well to begin at first by substituting only one or two bottles of the stronger mixture in order to see whether or not it agrees. In any case, it is advisable to make only one change at a time; therefore the bulk of the feeds and the composition ought not to be altered simultaneously. Between the sixth and eighth weeks it will probably be necessary to alter the diet again, this time giving a mixture of three parts of milk to one of water. Towards the end of the third month undiluted milk may be given. Throughout the whole process the quantities of milk given in the twenty-four hours should be checked by reference to the body-weight.

As an alternative to this method, many prefer to keep the milk-and-water mixture at the original strength, and to modify it by *adding cream*. Cream varies much in composition; gravity cream may be assumed to contain from 10 to 16 per cent. of fat. To add 1 oz. of this cream to 9 or 15 oz. of any milk-mixture is to enrich the total by 1 per cent. of fat. Thus, if 24 oz. of food is required for the day, a mixture of 11 oz. of milk, 11 oz. of water, 2 oz. of 12-per-cent. cream, and two level tablespoonfuls of sugar will contain approximately 2 per cent. protein, 3 per cent. fat, and 6 per cent. sugar. For those who prefer a slightly complicated to a simple method this plan may be recommended.

It is not proposed to give any of the usual quantities and proportions for different ages. Better results will be obtained if the needs of each baby are studied and the principles on which this method of hand-feeding should be carried out are borne in mind. They are: (i) Begin with comparatively low fat and protein, changing gradually to a stronger mixture. (ii) Use simple formulae, to lighten the mother's work and lessen the chance of mistakes. (iii) Be guided as to total quantities by the weight-curve, and as to size of feeds by age, allowing 1 oz. for each month. (iv) Avoid over-feeding; an infant does not usually need more milk daily than one-seventh of its weight.

Tolerance of milk is relative, and, though some infants can digest large quantities without trouble, others cannot do so. When the limit of tolerance is passed, difficulties arise. (v) Rich cream mixtures are bad; as a rule, 3.5 per cent. of fat is ample.

(b) *Feeding with undiluted milk.*—This is the simplest way of feeding infants, and in some ways the best. It was introduced by Prof. Budin of Paris, and most of the failures which occur are due to the improper carrying out of the method. The following rules should be strictly adhered to in feeding infants with undiluted milk:—

- i. It is only suited for babies who are free from digestive disturbance.
- ii. While the average normal infant usually supports undiluted milk from birth, in the case of premature, weakly infants it is safer to dilute the milk for the first fortnight.
- iii. The milk should be sterilized for 40 minutes at 212° F. in a Soxhlet or similar apparatus. It cannot be said that this *prolonged* heating is absolutely essential, but it has always given me such good results that I have never departed from it.
- iv. The feeds should be small. Neglect of this rule is the most common cause of failure.
- v. The quantity of milk given is controlled by the body-weight.
- vi. No sugar or addition of any kind is required.

In practice it is usual to begin with ten 1-oz. feeds daily. In a week the child is weighed, and, should it not have gained, the ration is increased—not otherwise. Large additions are not necessary: 2 or 3 oz. extra per day will usually suffice. The maximum daily allowance towards the end of the first year does not exceed 35 or at most 40 oz. The duration of sterilization should gradually be reduced to ten minutes.

It is scarcely needful to insist on the importance of scrupulous cleanliness in all that pertains to hand-feeding. The feeds should be warmed to blood-heat, and after the infant has finished a feed—twenty minutes being the maximum time allowed—the bottle should *at once* be rinsed and left to stand full of water. Before it is again used it must be cleaned thoroughly with a brush, soap, soda, and hot water. The teats should be rinsed after, and dropped for a minute into boiling water before use.

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Either boat-shaped or plain bottles are best ; those with tubes are inadmissible. An unfinished feed should be thrown away, and feeds should be warmed only immediately before they are required.

(5) **Common difficulties and minor ailments associated with artificial feeding.**—In many cases the food in itself is not to blame. Lack of cleanliness, irregular feeding, the custom of giving food from the family table in addition to the milk ordered, extremes of temperature leading either to chilling or overheating the body, and general lack of care, all lead to indigestion and failure to thrive, no matter how carefully the diet is adjusted. In approaching any case of dyspepsia or malnutrition, therefore, it is essential to correct any of these errors.

i. **Vomiting.**—The most common cause of vomiting is simple regurgitation from the overfilled stomach. This occurs shortly after a meal, before curdling has taken place. The remedy is obvious. In vomiting from dyspepsia the vomited matter consists of sour-smelling curd, and there are often also diarrhoea and colic. It occurs at irregular intervals. If the condition persists the diet must be modified. Begin by giving a dose of castor oil, and limit the quantity of food, especially the fat and sugar. Citrated milk sometimes answers well, or, especially in young infants, peptogenic milk.

ii. **Colic and flatulence.**—Gastric flatulence is often due to air-sucking. The remedy is to give the bottle carefully, so that the teat is kept full of milk, and not to allow the infant to go on sucking when the bottle is empty. Intestinal colic is very common ; the infant cries, draws up the legs as if in pain, and is relieved by the passage of flatus, often along with a loose, undigested motion. Some infants are very liable to such attacks, which are often brought on by chill. A full dose of castor oil is a prompt and effective remedy. If the attacks recur, such an alkaline carminative as the following is useful :—

Ry Sodii bicarb. gr. v.
Spir. ammon. aromat. ℥v.
Aq. ad ʒi.

Precautions should be taken against chilling, particularly of the legs and lower abdomen. The diet will require revision, and modification if necessary. Citrated milk often suits cases of this class. The pain of colic must be distinguished from that of fissure of the anus, and

from that of urinary colic due to the passage of uric-acid gravel.

iii. **Diarrhoea and constipation.**—The motions should be inspected in every case of digestive disturbance ; the amount of information obtainable, though limited, is often useful. Normal pale-yellowish motions of soft consistence, and free from visible mucus, show that the digestive organs are healthy. Abnormal motions indicate that the digestive tract is upset, but often it is impossible to go farther than this and to say in what particular respect the diet requires modification. Still, the following points may be noted :—

(a) Large, hard, crumbly, putty-like motions nearly always point to excess of cow's milk, and are an indication for diminishing the quantity taken, and giving some carbohydrate. This is one of the most useful hints derived from examination of the stools.

(b) Fat-diarrhoea is rather rare, but the appearance is characteristic. The motions are frequent, large, white, and pultaceous. They show that a great excess of cream is being given.

(c) Mucus may occur in the stools in any form of digestive disorder. The only mucous stools which are characteristic of a particular disease are the small, frequent evacuations, sometimes bloodstained, of dysenteric diarrhoea. They resemble those of intussusception. Such stools should be examined for bacilli of the dysentery group, and the patient ought to be looked on as infectious.

(d) Dyspeptic stools are fluid or semi-fluid, often greenish, and contain mucus and so called "curds." It is not possible to infer from these stools what particular constituent of the diet is at fault. The exact significance of "curds" is unknown. They appear to consist of soaps, as a rule, but sometimes contain masses of casein.

(e) Brown, frothy stools are indicative of carbohydrate fermentation, and do not occur in infants fed wholly on milk.

Slight attacks of *diarrhoea* should be treated by diluting the milk and limiting the daily quantity. If much sugar is being given, the quantity should be restricted. Skim-milk and water may be tried. At the outset a dose of castor oil should be given. After this has acted, bismuth is useful ; the following is a suitable mixture :—

Ry Bis. carb. gr. vii.
Pulv. cret. gr. iiss.
Pulv. trag. co. q.s.
Aq. ad ʒi.

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Another useful remedy in slight diarrhoea is the *mistura olei ricini*:—

R \bar{y} Ol. ricin. ℥x.
Syrupi ℥xv.
Muc. acac. ℥x.
Aq. chlorof. ad ʒi.
Ter in die.

If the diarrhoea persists notwithstanding these measures, milk should be discontinued and water only given.

In bottle-fed children constipation is often due to overfeeding, the motions having the appearance described above. The milk should be reduced, and half to one teaspoonful of a malted food, or malt extract, be added to each bottle. Much less commonly constipation is due to deficiency of fat, and can be remedied by giving cream. Habit is a factor in many cases; a good nurse will often succeed in training even a very young infant to evacuate the bowel at stated periods. The addition of fluid magnesia to the food is often a help.

iv. *Malnutrition*.—Bad marasmus—atrophy—is rare in well-cared-for children, and it is often incurable; slight grades of wasting, indicated by cessation in gain of weight, and flabbiness, are common, and usually respond fairly readily to treatment. The clinical features of slight malnutrition—"weight-disturbance," it is now sometimes called—are readily recognizable. A baby, hitherto thriving on cow's milk, ceases to gain but does not actually lose weight, and in place of being firm becomes soft and flabby. At the same time the bowels are confined, and the motions dry and putty-like. The history will show that the amount of milk taken has been ample, and often that increasing the quantity has made matters worse. There may be no digestive disturbance except the constipation. This is a common type of malnutrition, and its diagnosis rests largely on the history, and on the examination of the stools. The treatment is to diminish the amount of milk, and especially the milk fat, and to add carbohydrate. Thus one might order skim-milk, water, and a malted food, or in an older child an unmalted starch such as oat-flour. It is in this class of case that some knowledge of the caloric equivalents of different foods is of assistance. Another course, which is in some ways easier, and often satisfactory, is to replace the previous diet by a milk-and-malt proprietary food. Though many think otherwise, there is no doubt that such foods give excellent results in properly

selected cases. For the further management of marasmus, see *MARASMUS, INFANTILE*.

In all these digestive disorders the previous history as regards diet is very important, as it often affords the only clue to guide one in deciding which element of the food is to blame. The same symptoms may be produced by quite different foods, and it is a good rule in practice to begin by revising the former diet, especially as regards the relative proportions of fat and carbohydrate.

(6) *Modifications of milk and other substitute-foods*. *Diluents*.—Water is the best diluent. Barley-water is sometimes used with the object of forming a softer curd and giving some starch, but its advantages are doubtful. Alkalies are sometimes added; lime-water and bicarbonate of soda are the chief. The effect of adding an alkali is to delay rennin action, and to allow part at least of the milk to pass on into the intestine without undergoing gastric digestion. Lime-water also gives a peculiar mucoid appearance to the curd which forms. Alkalies are passing out of use; their value is more than doubtful.

Citrated milk.—Sodium citrate prevents the formation of curd, probably by precipitating the soluble lime salts. Half a grain to the ounce of milk diminishes, and 1 gr. almost abolishes, curd-formation. It is usually prescribed in a solution (to which 1 or 2 drops of chloroform are added as preservative) of the strength of $\frac{1}{2}$ or 1 gr. to the drachm; this is added to the feeds in the proportion of one teaspoonful to each ounce of milk. Citrated milk is undoubtedly useful in many cases of slight indigestion, and some infants can tolerate stronger mixtures of citrated than of plain milk.

Albumen water, made by shaking the whites of one or two eggs with 10 oz. of water, is the best substitute-food in diarrhoea. It is seldom advisable to restrict the diet to albumen water for more than four or five days.

Whey, prepared by adding rennin to milk and straining the curd (junket), and *wine whey*, made by boiling half a pint of milk with a wineglassful of cooking sherry, are sometimes employed during convalescence from diarrhoea to bridge the period between the cessation of albumen water and the return to the ordinary diet. They are now less used than formerly.

Buttermilk is useful in many forms of indigestion and malnutrition, especially when the previous diet has contained much fat. Ordinary buttermilk, not more than twenty-four hours

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old, may be used. To a pint and a half add 1 oz. of cane sugar and $\frac{1}{2}$ oz. of flour; boil, stirring vigorously to prevent lumpy curds forming. The caloric value of the mixture is about 17 $\frac{1}{2}$ Calories per ounce. Infants speedily become accustomed to buttermilk and take it well. Its value depends on its poverty in fat and its richness in carbohydrate. The casein, acidity, and bacterial content are unimportant.

Peptonized milk is most conveniently prepared by using peptogenic milk powder. Made according to the instructions given, the mixture is too rich in fat. The cream should therefore be omitted. Peptonized milk is an excellent food for young infants with whom plain diluted milk disagrees. Older infants do not always thrive so well on it.

Condensed milks.—These are chiefly of use when fresh milk cannot be procured. As temporary foods they are passable, but are not suited for prolonged use, as they may lead to rickets, and sometimes to scurvy. Condensed skim-milks ought never to be used. Condensed full-milks are either sweetened or unsweetened; the following examples show their composition:—

	Protein	Fat	Lactose	Cane sugar
IDEAL . .	8.3	12.4	16	—
NESTLÉ'S .	9.7	13.7	15	37.2

As these milks should only be used temporarily, the sweetened variety is not disadvantageous. A mixture of one full teaspoonful of Nestlé's milk to 3 oz. of water (the strength generally used) contains about 2.5 per cent. of fat and 1.8 per cent. of protein. Condensed milk is more easily digested than plain milk, and may be given instead of peptogenic milk when the cost of the latter is a consideration.

Dried milks differ from unsweetened condensed milk only in the fact that the process of desiccation is complete. Glaxo, one of the best-known, contains 22 per cent. of protein, 27 per cent. of fat, and 41 per cent. of sugar. They are prepared for use by mixing with warm water.

Lactalbumin.—Soluble milk protein is now marketed under several trade names. The idea underlying its use is (a) to increase the soluble albumin in the milk without increasing the casein, and (b) to prevent the casein already present from forming a tough curd. The theoretical justification for using lactalbumin is not very strong, and it is of no value in infant feeding.

Proprietary foods.—These are very numerous, but, in spite of individual difference, all fall into three main groups: (a) milk and malted starch; (b) malted starch; (c) unmalted starch. In some, malting is complete; in others it becomes so in the process of preparation for use. As types the following may be cited:—

	Protein	Fat	Carbo- hydrate
ALLENBURY No. 1 . .	9.7	20	60
MELLIN'S . .	6.3	7.9	82
SCOTT'S OAT FLOUR . .	5.8	9.7	78

Patent foods are not nearly so good for a healthy baby as cow's milk, and ought not to be substituted for it except in special circumstances. They have, however, a definite field of usefulness, and if properly employed are of value.

The unmalted starches (c) are not suited for children under 7 months old, but after this age, when carbohydrate is required, they are convenient (*see below*).

The essential feature of groups (a) and (b) is their richness in maltose and dextrins, which make up something like 80 and 20 per cent. respectively of the total carbohydrate. The value of maltose in infant feeding depends on the fact that it is less liable to ferment in the bowel than either milk or cane sugar. As pure maltose is very expensive, it is usually given to children either as malt extract or as one of these malted foods. Foods of the plain maltose-dextrin group (b) may be given instead of milk sugar in cases of dyspepsia—a combination which is not seldom useful in atrophic dyspeptic babies; they consist of skim-milk and water with 2 or 3 per cent. of maltose-dextrin. Foods of the dried milk-maltose group (a) are useful in cases in which there is intolerance of cow's milk. It is often desirable in using foods of this description to check by the caloric value the quantities given.

On any diet other than human milk an infant may develop scurvy, and it is especially liable to do so on a food containing much carbohydrate. This risk should be guarded against by giving some grape- or orange-juice daily if a diet of this kind is continued for more than a month or so.

4. *Wet-nursing.*—Wet-nursing is the best substitute for maternal nursing, but wet-nurses are not very easy to obtain in this country. A wet-nurse should be from 25 to 35 years old; the nutrition of her child is the best index of her milk supply. She should be clean, free from skin eruptions, and organically sound, and

should undergo the Wassermann test. Her own infant need not be of the same age as the foster-child, but should be from 3 to 6 months old. Steps should be taken to ensure that it is properly cared for, or, if possible, the wet-nurse should be allowed to suckle it as well as the foster-child.

5. Premature and weakly infants.—A premature or weakly infant should be fed on the same lines as a full-term baby, the feeds, however, being smaller and given at somewhat shorter intervals. Breast-feeding is especially important in such cases. If the infant is too feeble to suck, the mother's milk should be withdrawn by a breast-pump and given to the child from a spoon or dropping tube. Maintenance of the body-heat is essential. If an incubator is not available, a good substitute may be made by placing a fracture cradle covered by a blanket over the child as it lies in its cot, and keeping a 16-c.p. carbon-filament lamp burning under the cradle. By this means the air round the child is kept at a fairly constant temperature of 90°–100° F.

6. Weaning, and feeding after infancy.—The process of weaning from the breast or bottle ought to begin about the seventh or eighth month. The breast-fed child should be given one or two feeds of cow's milk diluted with water instead of the corresponding nursings. After a week the number of breast-feeds should be again reduced, and at the same time some starchy food given, as in the case of an artificially-fed child of the same age. Weaning should be complete in about a month.

When a hand-fed baby is about 7 or 8 months old, starch should be added to the diet. Oat-flour gruel is a convenient form, one or two tablespoonfuls being added to each bottle. In another month one of the bottles is replaced by a saucerful of farola or gruel, and towards the end of the year boiled bread-and-milk should be given.

When the child is a year old the yolk of an egg rubbed up with bread-crumbs is taken, and a little later the whole egg, thin bread, and a cup of milk form a suitable meal. Bread-and-butter is also allowed. The following will serve as a sample of the dietary for a child of 12–15 months: 7–8 A.M., bread-and-milk; 10–11 A.M., porridge milk; 1–2 P.M., egg, with half a slice of bread, and milk; 4–5 P.M., saucer of farola; 7–8 P.M., cup of milk.

From the eighteenth month a little fresh vegetable, potato and gravy, mashed cauliflower, and fruit such as banana, orange, or ripe

apple, may be given from time to time. During the latter half of the second year, bread soaked in bagon fat, thin soups, custards, jellies, fish, and occasionally a little roast mutton or lamb, or chicken, or chop, or mince, are suitable. Thus the following diet might be ordered: 7–8 A.M., milk, bread-and-butter, porridge, or egg; 10–11 A.M., cup of milk, or a banana if desired (usually better omitted); 12.30–1.30 P.M., minced or shredded chicken, meat, or fish, or potato and gravy, with bread-and-milk or custard, or a roast apple.

Just as in the first year of life it is a common mistake to think that milk is insufficient, and to add starches too early, so during the second year it is the custom unduly to restrict the diet, especially as regards animal food, and to give too much pappy food. On a diet of this kind starch indigestion is liable to develop, under-nutrition is not uncommon, and the habit of proper mastication is not properly formed. This last, according to dentists, is one of the chief causes of dental caries. There has doubtless been a good deal of exaggeration as to this, but it is certainly desirable to teach children to chew tough articles of food as early as possible. Chicken bones, hard crusts smeared with butter, and hard fruit such as apples, are best for this purpose. Some children take to them readily; others do not. The chief point, probably, is to ban eating sweets after a meal, and especially at bedtime, and to give fruit as the last food of the day, so that all fermentable starch and sugar may be removed from the interstices of the teeth by the cellulose of the fruit, and a flow of alkaline saliva be promoted by its acids.

During the later years of childhood the diet should be abundant, simple, yet varied, like the diet of the adult. Overloading the stomach with starchy food should be avoided, and the practice of boiling all milk for drinking should be continued.

J. S. FOWLER.

INFANTICIDE.—The crime of infanticide is usually committed immediately or shortly after the birth of a child. The child may be destroyed (1) by an act of omission, e.g. neglecting to tie the cord, or suitably to clothe and feed the child, or (2) by an act of commission, e.g. strangulation, wounding, etc.

To prove a charge of infanticide it is necessary to show not only that the child has lived, but that it was "born alive" in the legal sense. Owing to the difficulty in many cases of proving live-birth, the indictment usually includes an

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alternative charge (a) "concealment of birth" in England, or (b) "concealment of pregnancy" in Scotland.

A child is considered live-born in the legal sense when, after entire delivery from the parts of the mother, it has maintained a separate circulation or life of its own. The difficulty of proving this is in many cases great, and the law starts with the assumption that every newborn child has been born dead, and places on the prosecution the onus of proving live birth.

Among the questions which demand an answer in charges of infanticide, the most important are these :

1. Has the woman charged been recently delivered ?
2. Is the child found that of the person accused ?
3. What degree of maturity has the child reached ?
4. Was the child born alive ?
5. If born alive, what caused its death ?

Questions 1 and 2 are usually answered without medical evidence, but it should be remembered that if a medical man is asked to examine a woman in order to determine if she has recently given birth to a child, her consent to examination must be obtained after she has been warned as to the use which may be made of the information gained. There is no law to compel a woman to submit to such an examination.

3. The **degree of maturity** which the child has attained is important, as the chances of natural death are increased in proportion to its immaturity. The question of infanticide is not, however, influenced by the "viability" of the child (i.e. its fitness to continue life), as the destruction of a new-born, live-born child is murder. It may be taken as a general rule that few children born before the seventh calendar month are capable of surviving. The following data will assist in determining the maturity of the child :

At the end of the seventh month: Length 13-15 in., weight 3-4 lb.; nails not reaching to the end of the fingers, the pupillary membrane disappearing, the testes at the abdominal ring.

At the end of the eighth month: Length 15-17 in., weight 4-5 lb.; nails practically reaching the extremity of the fingers, pupillary membrane absent, testes in inguinal canal or one (usually the left) in the scrotum.

At the end of the ninth month: Length 18-24 in., weight from about 6½ lb. upwards; nails projecting beyond the tips of the fingers,

testes in scrotum, a well-marked node of ossification about one-quarter of an inch in diameter in the lower end of the femur. This should always be looked for in making a post-mortem examination. After removal of the soft parts covering the lower end of the bone, transverse slices are removed from the cartilaginous epiphysis until the ossific node (a red spot of gritty consistence) is found.

4. **Was the child born alive ?**—The child may be so immature as to have been incapable of separate existence; or there may be present malformations incompatible with life for more than a few minutes or hours—e.g. acephalous and anencephalous monsters, extroversion of the heart. The body of the child may not be found for some time after death, and putrefactive changes will have set in, and should not be diagnosed as due to intra-uterine maceration.

When the child has lived for some days there may be no difficulty in deducing live-birth from the umbilical cord having separated, but as a rule evidence of live-birth will only be derived from a post-mortem examination.

Evidence from the lungs.—When respiration has not occurred the lungs are small, firm, non-crepitant, and uniformly dark-reddish-purple in colour. If respiration has been established the lungs more or less fill the pleural cavities, are elastic and crepitant to the feel, and "marbled" in appearance.

Any undistended or atelectatic portion of the lung will retain the foetal appearances. Confirmation of respiration is obtained by the *hydrostatic test*, carried out as follows :

Remove all the organs of the thorax *en masse* and place them in a vessel of water, and note if the lungs are buoyant enough to support the weight of the heart and the thymus gland. Next detach the lungs and test the flotation of each lung separately; then cut each lung into twelve or more pieces and test each piece separately. Finally, wrap the pieces in a cloth and place them on the floor and subject them to pressure with the sole of the boot and again test their flotation. If the lungs give positive results under all these conditions, then the child must have fully and freely respired.

Objections urged against the hydrostatic test are :

(1) The lungs, or portions of them, may sink though the child has breathed, owing to disease (e.g. pneumonia) or persistent atelectasis. The gross changes of disease usually present no difficulty. As regards atelectasis, cases have been reported in which this condition has been found post mortem where there has been

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definite proof of breathing. Various explanations, such as the possibility of respiration through the trachea and bronchi, have been offered, but the only important point in infanticide is that the fact of atelectasis has been established.

(2) The lungs, or fragments of them, may float although the child has not respired. (a) *From putrefaction.* In this connexion it should be remembered that putrefaction of the lungs will be accompanied by putrefaction of all the soft parts of the body. Pressure as described under the test will expel the gases of putrefaction and the lung will thereafter sink. If putrefaction is far advanced, then pressure may expel the alveolar air as well as the gases of decomposition. (b) *From artificial inflation.* Lungs artificially inflated will be uniformly bright-red in colour, and on section there will be an absence of bloodstained froth. The evidence of the person who performed artificial respiration will usually be available.

Evidence from the alimentary canal.—The presence of food in the stomach may be taken as satisfactory evidence of live-birth, though its absence will not necessarily indicate still-birth.

Evidence from the umbilical cord.—An inflammatory line of separation makes its appearance about thirty-six hours after birth, and in the majority of cases separation occurs on the fourth, fifth, or sixth day.

Evidence from the circulatory system.—Owing to the variable period at which the foramen ovale, ductus arteriosus, and ductus venosus close, any evidence derivable from their non-closure is unreliable.

5. **What was the cause of death?**—Death may result from natural or accidental causes, as well as from violence. The violent causes may be any of those which occur in the case of adults, and the same questions will arise. Suffocation, strangulation, wounds and injuries to the head, etc., drowning, and burning are the methods most frequently employed. Poisoning is employed very rarely. In all cases of alleged infanticide special search should be made for the presence of foreign bodies in the air-passages. A defence is frequently set up that violence was caused accidentally during or immediately after parturition, and all the points must be weighed in coming to a decision. Thus the child may have been smothered in the bedclothes or the clothes of the mother in cases where delivery occurred without assist-

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ance being available. Where marks of manual strangulation are present a defence of attempted self-delivery is often set up, whereas a circular mark on the neck may be alleged to have been produced by the umbilical cord. When the cord has become coiled round the neck with fatal results, death takes place before respiration has been established, and from arrest of the circulation. The cord, however, might be used to strangle a child after it had breathed. Fractures of the skull have occurred in utero by accidents to the mother during parturition in tedious labour, or the child may have fallen on to the floor in precipitate labour. The history, the size of the mother's pelvis relative to the size of the child, whether the mother is a primipara or a multipara, the condition of the perineum as regards laceration, the distribution of the injuries, whether the umbilical cord is torn or is still attached to the placenta and child, the presence and size of the caput succedaneum—all these circumstances will assist in coming to a conclusion. Drowning has taken place by the child being precipitated into a water-closet or some other receptacle when the mother had or was attempting a movement of the bowels,

A. ALLISON.

INFANTILE CONSTIPATION (see CONSTIPATION, INFANTILE).

INFANTILE CONVULSIONS (see CONVULSIONS).

INFANTILE DIARRHŒA (see DIARRHŒAL DISORDERS OF INFANTS).

INFANTILE MARASMUS (see MARASMUS, INFANTILE).

INFANTILE PARALYSIS (see POLIO-MYELITIS, ACUTE).

INFANTILE SCURVY (see SCURVY).

INFANTILISM.—Abnormal stunting of physical stature is divisible into two groups of cases:—

1. **Infantilism (asexual ateleiosis).**—In these cases, with the retarded general physical growth there is delayed or absent development of the secondary sexual characteristics of puberty. The patient is, as far as physical development goes, persistently or permanently a child.

2. **Dwarfism (sexual ateleiosis).**—The patient here is more correctly described as physically a miniature adult. The changes of puberty, however, are usually delayed, and full procreative power is exceptional.

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The second group need not detain us longer ; it is with the first group that we are here concerned.

Etiology.—Formerly the various types of infantilism were differentiated by their physical characteristics, a descriptive classification. Thus Brissaud's type, Lorain's type, etc., were spoken of. With further progress in knowledge it is now possible to group the cases into classes according to their etiology, a symptomatic classification. Thus we speak of thyroid infantilism, renal infantilism, etc., meaning that the thyroid or the kidney is responsible for the retarded growth. There are still, however, some cases not yet fitted into any group, which can only be classed as instances of asexual ateliosis, the name suggested by Hastings Gilford, the substantive meaning "not arriving at perfection."

It may be regarded as a general rule that where an important organ of the body is diseased sufficiently seriously, at a sufficiently early age, retarded growth will occur.

In some instances it is obvious which organ is diseased, and the infantilism is of secondary interest. Thus, when the heart is severely affected congenitally or in early life, retarded growth is the rule. We may call this cardiac infantilism if we will. Similarly, infantilism may be associated with severe pulmonary disease, hepatic cirrhosis, inherited syphilis, or mental deficiency (e.g. mongolism, microcephaly).

In other cases the infantilism is the more apparent departure from the normal and the organ at fault less easy to recognize. These types will be described below.

Symptomatology.—The special types have their own special symptoms which will be described later. Here may be mentioned certain general symptoms common to all cases. There is retarded physical growth, with delay in the completion of ossification and deferred development of secondary sexual characters. The patients' weights are comparable to their heights, rather than to their ages, but often are lower than even this scale would allow. Mentally they remain children until puberty is reached, if ever it be reached. Mental deficiency of the true type is no part of infantilism. The patients are childish for their age, precocious for their appearance. The mental condition is best described as one of "experienced childhood." Delayed rickets, starting about the seventh year, has been seen in some of the special types of infantilism.

SPECIAL TYPES OF INFANTILISM

1. Mention has already been made of the types **secondary to gross organic disease**, or gross maldevelopment. The following four types may be recognized :—

(i) **Cardiac infantilism**, from severe congenital or acquired heart disease.

(ii) **Pulmonary infantilism**, from severe lung disease in early life.

(iii) **Syphilitic infantilism**, from congenital syphilis.

(iv) **Mental deficiency with infantilism**, from many forms of mental deficiency, e.g. mongolism, microcephaly.

2. We have now to describe cases of infantilism in which the **lack of growth is the most apparent defect**, and the origin of it less obvious than in the foregoing group.

(i) **Thyroid infantilism (hypothyroidism).**—The influence of the thyroid gland in promoting growth is well known. While the administration of thyroid will stimulate growth in some states not obviously dependent upon hypothyroidism, there are special cases of infantilism directly due to defective thyroid secretion.

The lesser grades of thyroid infantilism are probably those previously known as Brissaud's infantilism, characterized by large heads and rounded fullness of the soft parts. The severe and common grades are those of true cretinism, in which the stature may be extremely stunted and the mental deficiency of a serious type. It must be admitted that in many such cases thyroid administration produces very little benefit. Many instances of such incurable cases may be seen in any institution for the mentally defective. (See CRETINISM.)

(ii) **Celiac infantilism.**—This well-defined type is described and its treatment discussed under MORBUS CELIACUS (q.v.).

(iii) **Pancreatic infantilism.**—Many cases which have been described under this title almost certainly belong to the celiac group. The theory that pancreatic defect was the origin of the condition is probably wrong. It is, however, possible that infantilism may be associated with pancreatic disease acquired in early life, producing steatorrhœa of pancreatic origin ; such cases have been reported by Bramwell.

(iv) **Hepatic infantilism.**—This term was used for cases which would now be put in the celiac group. But in rare instances infantilism has been described as resulting from cirrhosis of the liver in children, and such

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cases would properly be described under this heading.

(v) **Renal infantilism.**—Another definite type of infantilism is associated with excessive secretion of urine and excessive thirst. These three symptoms, infantilism, polyuria, and polydipsia, are found in two forms of cases which together make up the renal group; in one organic renal disease is present (chronic interstitial nephritis), in the other it is absent (diabetes insipidus).

(a) *Renal infantilism with organic renal disease.*—This is the commoner of the two forms. Either from birth or from early life the symptoms of polyuria and polydipsia are found. The thirst may be so marked as to cause the child to drink the dirtiest water. The urine is of very low gravity, showing a faint trace of albumin on careful testing, but casts are very difficult to find. The cardio-vascular system may be severely affected. The left ventricle of the heart may be much hypertrophied, and the arteries, particularly the brachials, very definitely thickened. Curiously, other cases show no perceptible changes of this sort, even when death has occurred from uræmia and the kidneys are found extremely contracted. Pathologically, chronic active interstitial nephritis is found, of which the cause is unknown. The condition is not due to syphilis.

The infantilism here is usually of extreme grade, the stature of only half the true age being reached in some instances. As a rule, the patient is very pale, and shows a dry wrinkled skin and rather deep-set eyes. Late rickets, particularly knock-knee, is common after about the age of six or seven. The retardation of growth may be assumed to be due to the excessive wastage through the kidneys. Life is rarely prolonged after the twelfth year, death being commonly due to uræmia or pneumonia. I have seen more than one case come under observation for the first time for terminal convulsions and coma, due to this condition. *Treatment* is merely symptomatic.

(b) *Renal infantilism without organic renal disease.*—These are rare cases, really such as are usually classed as diabetes insipidus. The same symptoms are present as in the organic group, but the infantilism is not so severe, there are no cardio-vascular changes, the urine is free from albumin, and life is more prolonged. Some of the cases are associated with inherited syphilis, or with pituitary or other

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lesions of the nervous system (*see* DIABETES INSIPIDUS).

(vi) **Pituitary infantilism** (*hypopituitarism, dystrophia adiposogenitalis, Frölich's syndrome*).—The most conspicuous feature associated with the infantilism in these cases is adiposity. (*See* PITUITARY GLAND, AFFECTIONS OF.) Mention has already been made of the possible connexion of pituitary disorder and renal infantilism with diabetes insipidus.

(vii) **Adrenal infantilism.**—Cortical hypernephromata usually give rise to sexual precocity with early development of pubic hair and growth of genital organs. Two types are recognized: one with excessive muscular development (the "infant Hercules" type), the other with obesity such as has been described under (vi). In the former there is invariably sexual precocity, but in the latter this is sometimes replaced by genital hypoplasia (infantilism). (*See also* SUPRARENAL GLANDS, AFFECTIONS OF.)

(viii) **Infantilism from disease of other ductless glands.**—This type is not yet well recognized. Isolated cases of tumours of the testis, ovary, and pineal gland giving rise to the same symptoms as cortical hypernephromata have been described.

3. **Infantilism of unknown origin.** These cases, at present termed simply asexual ateleiosis, form but a very small proportion of examples of infantilism. They cannot as yet be classified.

REGINALD MILLER.

INFECTIOUS JAUNDICE (*see* JAUNDICE).

INFECTIVE DIARRHŒA (*see* DIARRHŒAL DISORDERS OF INFANTS).

INFECTIVE PSYCHOSES (*see* CONFUSIONAL INSANITY).

INFLUENZA.—An acute infective disease presenting varied clinical features, but showing a special tendency to affect the respiratory tract, which appears in world-wide epidemics at irregular intervals, and spreads with great rapidity.

Etiology.—There is some difference of opinion as to the causal organism. According to the more generally accepted view, Pfeiffer's *Bacillus influenzae* is the primary infective agent. This organism is frequently associated with others, such as pneumococci and streptococci, which appear to play the part of secondary invaders. On the other hand, it has been held that *B. influenzae* is but a

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secondary invader itself, the disease being primarily due to an ultramicroscopic, filter-passing organism. In the years following a pandemic, influenza shows itself for a time in an endemic or sporadic form. In these inter-epidemic periods especially, the term influenza is often applied without sufficient discrimination to trifling febrile ailments, the cause of which in any given case is not apparent. Future investigation will show that many of these cases have no connexion with influenza; some are probably the result of subminimal infections with the organisms responsible for the common zymotic diseases, others the result of infection of the respiratory tract with organisms producing the common cold. The term "influenzal cold," as applied to the latter type of case, is certainly less objectionable than the cognomen "influenza," and the expression has now passed into such common usage that it will be difficult of eradication. Many of these "influenzal colds" are due to infection of the respiratory tract with *Micrococcus catarrhalis*, *B. septus*, *pneumococcus*, *staphylococcus*, and other organisms. The correlation of the many types of common cold with their respective causal organisms lies within the field of investigation of the well-equipped general practitioner of the future.

Symptomatology.—The incubation period is short, two days as a rule, but it may vary from a few hours to four or five days. The rapid spread of the disease in times of epidemic is to be attributed to the shortness of the incubation period and the almost universal susceptibility of the population. There is little doubt that the path of infection is by way of the respiratory tract. In the epidemic of 1918-19, men who had been "gassed" in France proved more susceptible than their fellows, and the period of incubation was the minimum in these cases. The naso-pharynx is probably the portal of entry in the majority of instances. It is interesting in this connexion to recall the frequency with which epistaxis occurs as an initial symptom. Influenza may assume such differing clinical manifestations that for descriptive purposes the following clinical types will be considered: (1) The simple febrile, (2) the severe respiratory, (3) the abdominal, (4) the cerebral or nervous type.

1. **The simple febrile type.**—The onset is usually abrupt. A person in his usual state of health is suddenly prostrated with severe pain in the head, back, and limbs. There may be an initial chilliness, epistaxis, or vertigo. At

first the skin is hot and dry, but later there is often profuse sweating. The face is flushed, the conjunctivæ suffused, and slight drooping of the eyelids is not infrequently noticed. The temperature quickly reaches 103° or 104° F. The pulse does not show acceleration to the same extent, and commonly registers from 90 to 100 beats per minute. The tongue is tremulous and coated with fur, and the breath fetid. The throat is sore, making speaking and swallowing unpleasant; the voice is husky, and the throat is continually being cleared. Examination discloses reddening of the posterior part of the palate and of the fauces and pharynx, but it is exceptional to find any exudation or any swelling of the tonsils. Enlargement of cervical glands is infrequent, but sometimes there is tenderness below the angle of the jaw. Either there is no cough, or the cough is dry, irritable, and persistent, such as is seen in cases of tracheitis. Examination of the chest and abdomen usually fails to discover any abnormal signs, but in some cases there may be impaired resonance over the bases of the lungs, with feeble respiratory murmur, and exceptionally slight enlargement of the spleen may be detected. The patient complains of severe headache, located in the frontal region and at the back of the eyes. The pain is increased by coughing, by change of position in bed, or by movement of the eyes. He says that he aches as if bruised all over, and finds it difficult to obtain comfort in any position. Frequently the muscles are tender and the skin hyperæsthetic. Many patients sleep well, and merely desire to remain undisturbed, but in others sleep is fitful and slight delirium may appear. The appetite is lost, and cooling drinks alone are desired. The temperature falls on the second, third, or fourth day, and symptoms disappear by the end of the week. Nevertheless, the simple febrile form may develop into any of the other types, and it is wise to regard any case as potentially grave, since pulmonary complications may arise with great suddenness, even in mild cases. Patients are left weak and prostrate for a time, but in most cases are able to resume work within fourteen days.

2. **The severe respiratory type.**—The disease is ushered in with the same symptoms of fever, prostration, and aching pains as in the simple febrile type, but cough and respiratory embarrassment soon become prominent. The patient generally complains of pain in the chest, worse on taking a deep breath, or of a sensation

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of oppression and rawness behind the sternum. The cough is short, dry, especially troublesome at night, and may come on in paroxysms continuing for an hour or more at a time, distressing and exhausting the sufferer exceedingly. After a time, secretion is established and the cough becomes purposive, but the sputum is often very scanty and tenacious. Not infrequently it is frothy at first, and becomes purulent after some hours, or it may be mucoid and bloodstained from the beginning. In some instances there is profuse hæmoptysis. Blood-examination usually reveals the existence of leucopenia.

The clinical features of the case will vary with the pathological lesion which is present. When the inflammatory change is confined to the larger and the medium-sized tubes, the symptoms are milder than when extension to the smaller tubes and pulmonary parenchyma has taken place. Likewise, the physical signs will vary with the pulmonary lesion. They may be simply those of extreme bronchitis with dry pleurisy, and may be so pronounced as to mask any sign of consolidation should it be present. Even when a definite influenzal pneumonia develops, there is rarely any complaint of breathlessness, which is the more surprising since objectively the respiratory rate may be accelerated to 50 or 60 a minute. Orthopnoea, too, is exceptional. The pulse-rate increases with the temperature, but not to a corresponding degree. Arterial tension is low and the pulse frequently dicrotic. On examination of the chest, the physical signs at first may be slight in proportion to the intensity of the symptoms, but sooner or later evidence of consolidation usually appears. Contrasted with a frank lobar pneumonia, the physical signs are ill defined. Percussion reveals relative impairment of resonance, but any areas of defective resonance are much less distinct and their limits less clearly marked off than in an ordinary lobar pneumonia. The breath-sounds are noticeably weak, especially over the affected portions of the lungs. By making the patient cough, high-pitched, hissing, or sticky râles can often be elicited over areas where diminished breath-sounds were previously alone audible. Indeed, râles are detected much more frequently than bronchial breathing, which rarely reaches the development so characteristic of a croupous pneumonia. Another remarkable feature is the variation of the physical signs from time to time. This is probably to be accounted for by the occurrence of a temporary

collapse of certain areas of lung, by the rapid pouring out of a thin inflammatory exudate into the pulmonary parenchyma (an active œdema), and to a less extent by clearing of the bronchial tubes by a bout of coughing. Thus weak, but definite, bronchial breathing, accompanied by consonating râles, may be heard over a considerable area of a lower lobe, let us say, while later in the same day the signs suggestive of a consolidation may have disappeared entirely, the breathing being vesicular in character and the râles possibly replaced by rhonchi. Friction is to be heard in many cases, though unless special pains be taken to induce the patient to draw a sufficiently deep breath it is likely to be overlooked; it is perhaps most frequent in the postero-inferior portion of the axilla. Apical consolidation is exceptional, but appears to be more common than in ordinary lobar pneumonia. Albuminuria, though not necessarily indicative of nephritis, is much more frequent in influenzal than in pneumococcal pneumonias.

In the most severe cases anoxæmia sooner or later manifests itself by the appearance of cyanosis, which was commonly described in the epidemic of 1918-19 as heliotrope cyanosis. This sometimes occurs as early as the second day of the illness, and, once it has appeared, the patient is almost certain to die. In a naturally pale person it is most noticeable in the lips and ears, but in the more florid the whole face assumes a heliotrope colour. In the majority of these cases the pulse does not point to cardiac failure as the likely cause. It has been suggested that the anoxæmia is the result of interference with the normal gaseous interchange between the air and the pulmonary capillaries by the presence of a rich albuminous exudate which fills up large numbers of the alveoli and forms an impermeable medium to the passage of oxygen. Death may occur within twenty-four hours of the first appearance of cyanosis, or be delayed for days. Venesection commonly fails to relieve, or relieves temporarily at the most. Though so extremely ill, the patient appears to be unaware of the gravity of his condition, and may remain fully conscious and mentally alert till within half an hour or so of death. In other cases a rambling delirium with subsultus and much picking at the bedclothes precedes the *exitus letalis*.

The course of the temperature corresponds to the pulmonary lesion present. Some cases terminate by crisis about the eighth or ninth

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day; in others the fever ends by lysis, the temperature taking eighteen to twenty-one days to reach normal; but many intermediate modes are seen. In many cases, besides albumin a little blood is present in the urine, but rarely enough to produce a red colour. Speaking generally, it may be said that a patient with influenzal pneumonia is much more severely ill than one with an ordinary lobular or lobar pneumonia. In some of the less fulminating cases definite signs of over-distension of the right side of the heart make their appearance, and the patient, his skin bathed in cold perspiration, and his pulse growing feeble and more rapid and more intermittent, slowly sinks.

In quite a number of cases pulmonary complications have arisen at the end of a mild febrile attack of influenza after the temperature has subsided. Many of these are probably to be attributed to the patient's imprudence in getting about before the infecting organism has been completely got rid of. Occasionally cases are seen which are perhaps more correctly described as septicæmic rather than respiratory in type. They prove no less fatal than a septicæmic plague. Their condition is critical within a few hours from the onset; the heliotrope cyanosis makes its appearance, they develop all the appearances of the most intense toxæmia, and death may occur within twenty-four hours. In tropical countries such cases can be distinguished from pneumonia plague by the absence of *B. pestis* in the sputum.

3. **The abdominal type.**—In this form structural changes in the abdominal viscera comparable to those occurring in the respiratory organs rarely if ever occur. The symptoms appear to be those of disordered function, rather than those which depend upon structural alteration. The attack begins suddenly with abdominal pain, vomiting, and sometimes diarrhoea. The patient is prostrated, commonly complains of pain in the head and back, has complete loss of appetite, and is found to be running a temperature of 100° or 101° F. The tongue is furred, the breath foetid. Sore throat, and perhaps cough, help in indicating the true nature of the disease, which at first may be mistaken for some malady requiring surgical interference. In some instances the possibility that the case is one of typhoid may arise. Jaundice and parotitis are the complications more especially met with.

4. **The cerebral or nervous type.**—Here the headache, pain in the back, and prostration

which usually characterize the onset of the disease gain unusual prominence. Insomnia and delirium may occur during the febrile period, and the condition may be difficult to distinguish from a true meningitis. The term *meningism* is sometimes applied to this state, and there seems to be some post-mortem evidence for suggesting that it may be dependent upon œdema of the meninges. Mental and nervous exhaustion may appear after the patient's recovery, and neuralgia may be a troublesome sequela. All effort becomes irksome, desire fails, and no interest is felt in the ordinary activities of life. If unchecked, the condition may pass into definite melancholia. Among the more serious complications of nervous influenza must be included meningitis, meningo-encephalitis, myelitis, and peripheral neuritis; and cases resembling acute Landry's paralysis have occurred.

Complications. *Inflammation of accessory sinuses.*—Inflammation or even suppuration of the sphenoidal or ethmoidal sinuses is not uncommon; the frontal sinuses are more rarely affected. Deafness and pain in one or both ears during an attack usually points to the existence of otitis media; this is seen more commonly in children, and may go on to involve the mastoid cells.

Pleural effusion in limited amount is fairly common. It shows a special tendency to become purulent, though in many instances, where paracentesis is not delayed, complete absorption occurs. Pneumococci and streptococci are the organisms commonly isolated, and empyema is stated to occur in about 4 per cent. of cases.

Cardiac complications.—Acute dilatation of the heart, the result of a toxic myocarditis, may arise during an attack. In addition to displacement of the apex, with a feeble irregular pulse, there are pallor, extreme bodily weakness, and a liability to syncope. In the aged or debilitated, sudden death from cardiac failure occasionally occurs several weeks after apparent recovery from influenza. Pericarditis is rare. Infective endocarditis not infrequently appears to date from an attack of influenza. The results of blood examination indicate that infection is usually to be ascribed to secondary invaders like streptococci. Micro-organisms morphologically indistinguishable from influenza bacilli have been isolated from the vegetations in a number of cases. Tachycardia, heart-block, and other disturbances of rhythm are not very infrequent sequelæ of influenza.

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Nervous complications.—Meningitis is fortunately rare. Meningococci, the influenza bacillus, pneumococci, and streptococci are the chief causative organisms. Other nervous complications include encephalitis, myelitis, and peripheral neuritis.

Hæmorrhage.—The frequency of epistaxis has been noticed already. Less commonly, hæmoptysis, hæmatemesis, menorrhagia, metrorrhagia or hæmaturia may be met with. Hæmoptysis is certainly more frequent than in ordinary pneumonia. It appears to be due to an intense active congestion of the pulmonary capillaries; in a few cases it is to be attributed to the lighting up of a quiescent tuberculous lesion (see below). Hæmatemesis is usually the result of hyperæmia of the gastric mucosa, but, occasionally it is ascribable to the vomiting of blood which has reached the stomach after epistaxis. In some cases there is *hæmorrhage into the rectus abdominis*, with or without spontaneous rupture of muscular fibres. Coughing appears to be the immediate cause, but a necrotic change, allied to Zenker's degeneration in cases of typhoid, is the essential predisposing condition. Acute pain and localized rigidity are common phenomena, and, as it is the lower segments of the rectus that are affected, acute appendicitis may be simulated in some cases.

Spontaneous emphysema of the chest-wall.—Another phenomenon sometimes seen after coughing is cracking of the skin of the chest, neck, and abdomen. Probably a minute rupture of the wall of an air-vesicle or a softened bronchus provides a way of escape for the air into the peribronchial tissues, whence it reaches the subcutaneous tissues of the neck and chest by way of the hilum of the lung and the mediastinum. In most cases in which this condition develops, the patient dies.

Nephritis.—Though transient albuminuria is of common occurrence, nephritis, attended by œdema, is quite exceptional.

Parotitis.—This is probably the result of an infection from the mouth. One or both glands may be affected. In some cases the other salivary glands share in the inflammation, and it may be difficult to exclude the possibility of coexisting mumps. Where orchitis also exists, it is doubtful whether a diagnosis of mumps can be definitely negated.

Ocular complications include panophthalmitis, conjunctivitis, and keratitis.

Rashes.—Herpes, as in pneumonia, is sometimes seen. Erysipelas occasionally coexists. Erythematous rashes, like those of scarlet fever,

measles, or German measles, sometimes occur and may give rise to difficulty in diagnosis. Purpura is rare; if generalized it is of the gravest import.

Sequela.—Certain of these have been mentioned already among complications. Both *encephalitis lethargica* and the so-called *nona* coincide in their appearance with worldwide epidemics of influenza, and the relationship which these diseases bear to influenza, when elucidated, will form an interesting chapter in epidemiology. **Pulmonary tuberculosis.**—Tuberculous patients living under sanatorium conditions do not appear to be more liable than the healthy, but when sufferers from this disease are attacked by influenza the tuberculous infection progresses and the patients rapidly lose ground and weight. Influenza occasionally reawakens the disease in the apparently cured. Apical collapse occurring as a sequel of influenza has sometimes been mistaken for a tuberculous infiltration. Its temporary character will serve to distinguish it from the more serious disease. **Rheumatoid arthritis** may date from an attack of influenza. The latter disease, by lowering the patient's resistance, permits organisms lodged in some pre-existing focus of infection to make successful raids upon the body. **Bronchiectasis** is another serious sequela. In some cases *B. influenza* has been recovered from the sputum years after the original attack. Distressing *vertigo* from involvement of the labyrinth is sometimes encountered.

Relapses are frequent and re-infection is by no means rare, influenza producing but a short-lived immunity.

Pathology.—Apart from cases of primary cardiac failure, death rarely occurs unless some serious affection of the lungs is present. In the fulminating form where death takes place within 24-48 hours, the lungs may show little beyond intense congestion and œdema to account for the suffocating dyspnoea, though subpleural, subpericardial, and subendocardial hæmorrhages bear witness to the profound toxæmia. Where the patient survives more than two or three days, evidence of intense inflammatory change is obvious throughout the respiratory tract. The mucous membrane of the sphenoidal sinuses is injected, or the sinuses, together with the ethmoidal cells, contain pus. The lining of the trachea and bronchi is of a crimson colour, and sometimes presents a glazed appearance from the presence of a dried coagulable exudate. Thick muco-pus can be

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made to exude from the bronchioles, occasionally in large quantity. The pleura often shows the presence of a thin, yellowish, slightly granular pellicle very different from the thick buttery layer so frequently encountered in pneumococcic cases. Not uncommonly, turbid fluid varying from a few ounces to a pint or more is found within the pleural cavity. On peeling off the fibrinous pellicle, numerous subpleural petechiæ are visible, and groups of pale points glimmering through the pleura will indicate the existence of miliary abscesses.

The lungs are heavy from the presence of inflammatory products, and the lower lobes are usually the most extensively affected. On section, different appearances are met with corresponding to the intensity of the infection and the variety of organisms present. In the most common type, in which, in addition to the influenza bacillus, pneumococci and hæmolytic streptococci are frequently found, the section presents a conglomeration of morbid appearances. Patches of broncho-pneumonic consolidation alternate with bright-red strips of congested lung, while interposed in utter disorder are diffuse areas of hæmorrhage or wedge-shaped tracts of infarction. At other spots, areas of collapse lie cheek by jowl with patches of alveolar or interstitial emphysema, and such parts of the base of the lungs as are not otherwise metamorphosed exude fluid and pit on pressure in manifestation of passive œdema. Further scrutiny reveals the existence of a purulent bronchiolitis, and perchance groups of miliary abscesses, while an active inflammatory œdema, perhaps the most typical of all the morbid changes seen in an influenzal "pneumonia," becomes evident on histological investigation. Microscopical examination of areas of lung not definitely consolidated shows the presence of a peculiar homogeneous structureless material (staining feebly with eosin) completely filling up many alveoli and bronchioles and entangling, it may be, a few cells. Within this faintly staining medium, clear rounded cavities form a striking feature; the whole aspect has been likened not inaptly to Gruyère cheese. The influenza bacillus is perhaps most likely to be isolated from such a lung when cultures are taken from the hæmorrhagic areas or from the bronchioles. In a much smaller number of cases naked-eye section of the lung resembles at first sight a lobar pneumonia, differing in that the consolidated portion lacks the relatively dry granular surface so characteristic of a pneumococcic consolidation. Indeed

in the influenzal cases the surface is peculiarly shining and moist; this probably depends upon the presence of an excessive amount of inflammatory œdema. In such a patch of consolidation Goodpasture would regard the existence of greatly dilated terminal bronchioles exhibiting a peculiar hyalin lining as strong evidence of its influenzal origin. In other cases the chief lesion is a bronchiolitis with inflammatory changes largely confined to the surrounding alveoli (interstitial broncho-pneumonia). This condition is better appreciated by feeling than by sight. The lung, when handled, appears to contain large numbers of pea-like bodies, the intervening lung tissue being soft and non-resistant. On section of the nodule it may be possible to express pus from its centre. In these cases *B. influenza* is not infrequently to be obtained in pure or almost pure culture, and clinically the cases run a relatively long course (three weeks or so). In any of these varieties enlargement of the bronchial glands is usually evident. In the more acute cases the glands situated at the bifurcation of the trachea may be many times their natural size.

The heart-muscle is usually somewhat pale, and breaks down easily under pressure. These changes are dependent upon cloudy swelling or early fatty change. There is commonly some degree of dilatation of the ventricles, the right being more frequently distended than the left. The condition of the myocardium will account for the low-tension pulse so commonly encountered. Subpericardial and subendocardial petechiæ are not infrequent. The kidneys may show a mottled surface; on section, the cortex is swollen, and blood tends to ooze from the cut vessels; the parenchyma is easily friable, and hæmorrhages beneath the mucous lining of the renal pelvis are frequently seen. The suprarenals may show intense injection of the medullary portion, or focal necrosis. In some cases hæmorrhagic extravasation into the gland and even thrombosis of the suprarenal veins have been found. These changes, too, may possibly be related to the low-tension pulse frequently met with. The meninges may be œdematous. Multiple punctate hæmorrhages have been found scattered throughout the white matter of the brain when encephalitis has preceded death.

Diagnosis.—In the early stage of the disease, if no widespread epidemic is prevailing, it may be impossible to make a confident diagnosis. The abdominal form, when ushered in by diarrhœa and vomiting, may have to be dis-

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tinguished from so-called *ptomaine poisoning*. The existence of a subnormal temperature, the greater degree of collapse, and a history of the ingestion of questionable food are points in favour of the latter. The various forms of *colic* and *peritonitis* will be no less easily distinguished. Rashes resembling those of scarlet fever, measles, and German measles sometimes give rise to difficulty. The *scarlatiniform rash* differs from that of scarlet fever in not being punctiform, in varying in intensity from hour to hour, in being pleomorphic, and in not showing the typical distribution of the scarlet-fever rash. A history of a previous attack of scarlet fever may help, and in influenza the tonsils are unlikely to be swollen or overspread by exudate. In *measles* the rash appears on the fourth day of the disease, but in influenza the morbilliform rash does not make its appearance at any definite period. In *measles coryza* will dominate the picture in the early days; in influenza, the pain in the head, back, and limbs. From *German measles* influenza will be distinguished by the absence of marked glandular enlargement and the presence of severe malaise and prostration. In *typhoid* or *paratyphoid fever* the invasion is less rapid and the early symptoms are less severe than in influenza. In a doubtful case a fall of temperature and amelioration of symptoms on the third or fourth day will favour a diagnosis of influenza. If there is still difficulty, hæmoculture and agglutination tests will decide the question. Several distinctions between *primary croupous pneumonia* and influenzal "pneumonia" have been already noticed. In influenza, symptoms of fever and general malaise are often present for two or three days before definite signs of pulmonary consolidation show themselves, and pain in the body predominates over thoracic pain. The sputum, even if rusty, lacks the gelatinous consistency so typical of pneumonia; in cases of doubt, examination for the presence of *B. influenza* should be carried out. There is usually an absence of leucocytosis in influenzal pneumonia. During the war it was sometimes difficult to distinguish between influenza and *trench fever*. True shin pain is scarcely ever observed in influenza. Where a considerable proportion of a unit were affected simultaneously, it was safe to assume that the disease was not trench fever, since this disease does not show the intense epidemic character of influenza. *Poliomyelitis*, in its early stages, is not infrequently styled influenza, and the

resulting paralysis has sometimes been attributed to "influenzal neuritis." In some cases of influenza, rigors, profuse sweating, and an intermittent temperature may suggest the existence of *malaria*. A careful blood-examination will assist in cases of doubt.

Prognosis.—The character of the prevailing epidemic very profoundly modifies the prognosis. In many epidemics it is the very young, the aged, and the weakly who have succumbed, but in others, notably so in 1918-19, it was the strong young adult who was mortally struck down. By far the largest number of deaths is to be attributed to pulmonary complications. Broncho-pneumonia is always serious. Cyanosis, a respiratory rate of 40 or over, and high fever are unfavourable signs. The appearance of extensive pleuritic friction over a much wider region than the area of consolidation is of grave import. The development of local suppurating foci is on the whole favourable. A guarded prognosis is to be given when the patient is a chronic alcoholic, and in cases with pre-existent cardiac or pulmonary disease. The mortality among pregnant women is high.

Prophylaxis.—Although conclusive proof of the efficacy of vaccines in preventing influenzal infection is wanting, the trend of opinion is in favour of their employment. They are most appropriate where large numbers of persons are collected together, as in schools, training colleges, and barracks, and in the case of persons especially prone to the disease. A useful vaccine for prophylactic use is one containing 80 million streptococci, 200 million pneumococci, and 400 million *B. influenza* per c.c. Half a c.c. should be given for the first dose, and a full c.c. for the second, an interval of ten days elapsing between the two injections. Other preventive measures consist in the use of some simple disinfectant gargle (such as liq. sodæ chlorinatæ 20 min. in a tumbler of warm water) twice or thrice a day, and in avoiding public meetings and entertainments. Those in immediate attendance on persons suffering from influenza should wear masks.

Treatment.—Early resort to bed is to be advised in all cases, nor should the patient be allowed to get up till the temperature has been normal for at least four days and all adventitious sounds have disappeared from the chest. Numerous fatalities have followed the neglect of these several precautions. Whenever circumstances permit, the patient should be isolated. Cooling drinks and milk are all that

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he will require in the way of food in the first few days. For the pain in the back and limbs in the early stages, *sod. salicyl.* 15 gr., *pot. bicarb.* 15 gr., *ol. limonis* 1 min., *aq. chlorof.* to 1 oz., every four or six hours, usually affords relief, but should the paroxysmal cough be the chief cause of distress a sedative linctus may be substituted. Where there is much restlessness and insomnia, *pulv. ipecac. co.* 10 gr., with or without the addition of *trional* 10 gr., may be given with advantage for the first night or two. As soon as the pains are relieved and bronchial secretion is established, expectorants are of service, as, for example, *ammon. carb.* 3 gr., *pot. iod.* 3 gr., *tr. nuc. vom.* 10 min., *ext. glycyrrh.* liq. 20 min., *inf. cinchonæ* to 1 oz. Where the bronchial secretion is extremely viscid a glass of hot milk containing half a teaspoonful of bicarbonate of soda taken the last thing at night, by diminishing the viscosity of the secretion, will materially aid in its expectoration. Indeed, at this stage the use of alkalis, which loosen secretion and in consequence lessen the distress of coughing, is a sounder measure than the employment of sedative remedies which depress the cough reflex. Opium and its preparations should be avoided entirely, or only given with the greatest caution, if bronchopneumonia be present. Steam inhalations are useful in all cases with much bronchial or tracheal irritability, and the same may be said of fresh air. Where broncho-pneumonia develops, the treatment does not differ in essentials from that of an ordinary case of pneumonia. Warm applications to the chest (poultices, antiphlogistine, stimulating liniments) not only afford comfort but possibly help to loosen the bronchial secretion and indirectly allay cough. A mixture which was largely used in the late epidemic, and earned a well-merited reputation, consists of creosote $2\frac{1}{2}$ min., *pot. iod.* 5 gr., *sp. vini rectif.* 10 min., *ext. glycyrrh.* liq. 15 min., *syr. tolut.* 30 min., *aq.* to 1 oz., every four or six hours. Many physicians favour the use of digitalis or strophanthus as soon as any pulmonary consolidation can be detected. If cyanosis appears, the continuous inhalation of oxygen passed through warm water containing a little alcohol is worthy of trial. As a mask often produces a sense of suffocation, the oxygen is preferably administered through a catheter passed into the nostril. Good results have also been recorded following the intravenous infusion of hydrogen peroxide. Oxygen in a state of solution in the blood, as ensured by this method, appears not

only to benefit anoxæmia but also to counteract toxæmia. The solution recommended is 2 oz. of H_2O_2 in 8 oz. of normal saline, and fifteen minutes is allowed for the infusion, the flow being temporarily checked every few minutes or whenever a large bubble of oxygen appears in the cannula or the patient becomes restless. In toxic cases in which the heart is showing indications of failure the intravenous infusion of 250-300 c.c. of a 5-per-cent. or 10-per-cent. solution of glucose is sometimes quickly followed by improvement. The subcutaneous injection of 20 gr. of camphor dissolved in 1 dr. of sterile almond oil, repeated, if necessary, after twenty-four hours, may also be of use in such cases. Another plan that has been adopted in toxæmic cases is the subcutaneous injection of 1 c.c. of turpentine in the hope of producing a fixation abscess.

If effusion into the pleura takes place the special tendency to empyema must be remembered, and exploratory needling may well be carried out earlier than would be judged necessary in other circumstances.

If vaccines are used at all in the curative treatment of the disease, they would seem to be most appropriate for chronic cases. Encouraging results are reported where serum, obtained from a patient recently convalescent from a severe attack, has been injected subcutaneously during the acute stage of the disease.

During convalescence the diet should be generous, vegetable bitters like *nux vomica* or *cinchona* should be exhibited, and change of air and scene is very desirable. Especially should these directions be enforced if much nervous and mental exhaustion is manifest. Morphia should never be used for the relief of insomnia or neuralgia following influenza.

Lastly, reference must be made to the many forms of abortive treatment which have been employed. Salicin 20 gr. has been given every hour for twelve hours and then every two hours for the following twelve hours. Others report equally good results from the use of phenol $\frac{1}{2}$ gr. every hour or two till the urine becomes dark brown, an event which usually occurs within twenty-four hours.

C. E. LAKIN.

INFLUENZAL PNEUMONIA (see INFLUENZA).

INGROWN TOE-NAIL (see TOE-NAIL, INGROWN).

INHIBITION (see NERVOUS SYSTEM, PHYSIOLOGY OF).

INJURIES FROM THE MEDICO-LEGAL STANDPOINT

INJURIES, ABDOMINAL (*see* ABDOMINAL INJURIES).

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Any form of external or internal injury caused by violence is a *wound* in the legal sense. The term covers not only breaches of continuity of the true skin or mucous membrane—the incised, punctured, lacerated, and contused wounds of surgery—but also bruises, burns, fractures, and ruptures of internal organs.

To convey some idea of the danger to life resulting from a wound, the terms “slight,” “dangerous,” and “mortal” are employed in courts of law. A *slight* wound is one which either does not incapacitate or does so only for a short period, and will heal rapidly without leaving permanent incapacity. A *dangerous* wound is one which, on account of its extent or relation to vital organs, is a menace to life but is not necessarily attended by fatal consequences. A *mortal* wound is one which of necessity causes death immediately or within a short time of its infliction. The qualifying term should have reference to the primary condition produced by wounding, and not to a possible secondary cause of death, such as sepsis or tetanus.

Responsibility.—A man is responsible for the death of a person he has wounded if the death be directly or indirectly due to the violence and if it take place within a year and a day from the date of the injury. If an operation is necessary for the treatment of the injury and the injured person dies during or after the operation, the responsibility of the assailant remains if the operation was undertaken bona fide and was performed with reasonable care and skill.

Causes of death after wounding.—The cause of death after violence may be external or internal hæmorrhage, injury to vital organs, or shock (primary causes), septic infection, tetanus, exhaustion, delirium tremens, etc. (secondary causes). In fatal cases of wounding the actual cause of death should always be stated, so that it may be understood why death resulted from a particular injury; thus, “stab wound of chest” is not in itself a sufficient cause of death, and should be qualified by the addition of the actual cause, e.g. “and hæmorrhage from punctured wound of lung.”

Death from hæmorrhage.—The post-mortem signs after severe bleeding are general blanching of the body, pallor of the lips and mucous mem-

branes, faint post-mortem lividity, comparative emptiness of the heart and great vessels, and comparative bloodlessness of the internal organs, especially the spleen, liver, and kidneys.

The actual amount of blood which must be lost to cause death depends on the individual; a debilitated and anæmic person may succumb from a loss which would be recovered from by one who is strong and robust. Where hæmorrhage has occurred into a body-cavity such as the peritoneal cavity the amount of blood found should be accurately measured.

Death from shock.—Whereas shock is a common cause of death after severe injuries, it may also follow slight violence, e.g. after blows on the epigastrium or injuries to the testicles. No characteristic post-mortem appearances are present, and the diagnosis depends on the nature of the violence and the absence of other conditions sufficient to account for death. In such cases the heart should be examined carefully, as sudden death is common when there are pathological changes in it, such as fatty degeneration of the myocardium.

Injury to vital organs.—When there has been injury to the heart, lungs, or central nervous system, etc., careful note should be taken of the existence of any pathological condition, e.g. aneurysm of arteries, phthisical cavities in lungs. In such cases it will be necessary to decide (1) whether the violence by itself caused death, (2) whether death was due to the pathological condition alone, or (3) whether the violence contributed to or accelerated death. In some cases the answer is obvious, in others extremely difficult, as when a person dies from hæmorrhage into the substance of the brain after a slight blow on the head, and the cerebral arteries are found to be atheromatous and therefore liable to rupture without any external violence having been applied.

Points to be noted in cases of wounding.—To be able to answer the questions which will subsequently arise, the medical examiner should note the total number of wounds; the situation of the wounds relative to anatomical landmarks; the direction, dimensions (accurately measured), and character of the wounds, and the condition of the surrounding tissues; the presence of foreign bodies (e.g. glass, broken earthenware, etc.) in the wounds; the hæmorrhage, and the bloodstains on the body, clothing, and surrounding objects; the evidence of vital reaction and inflammatory processes. If there are cuts in the clothing, the articles should be examined singly and superimposed. A

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garment which has been folded upon itself may present two holes from a single stab wound.

Situation, dimensions, and direction of wounds.

—Suicidal wounds are usually found on parts within easy reach of the hand (generally the right hand in right-handed persons), such as the throat, chest, forearm, groin, and thigh. Wounds on the vertex of the head and the back are more likely to be homicidal or accidental. Multiple wounds most frequently indicate homicide, though they are sometimes suicidal or accidental. From the direction of a punctured or gunshot wound some idea may be got of the relative positions of assailant and assaulted.

By what kind of instrument was the wound produced?—The shape of the wound may bear no relation to the shape of the instrument which produced it, but often a study of the wound yields positive information. Its character—incised, punctured, lacerated, or contused—will usually enable the witness to state the kind of instrument by which it might have been produced (*see below*). Sometimes an assaulted person may present contused, lacerated, and incised wounds which have all been inflicted by the same weapon—usually a bottle, earthenware jug, or similar article. Contusions and lacerations are first produced, and then the bottle or other article breaks, and incised wounds are caused by the broken end.

Was the wound produced before or after death?—The points to be noted are any evidence of vital reaction, and the hæmorrhage—clots in the wound, the presence of sprays of blood, indicating that the blood had been forcibly projected from the cut end of an artery, and staining of the tissues of the wound by blood. Signs of vital reaction can never be observed in mortal wounds. In contusions signs of ante-mortem production are swelling of tissues and the presence of blood in subcutaneous cellular tissue as revealed by incision. At a post-mortem examination all marks of discoloration suspected to be the result of violence should be incised to differentiate bruises from staining due to hypostasis. In the latter case no blood will be found extravasated into the tissues, and only a few bleeding-points from cut capillaries will be observed.

Accident, suicide, or homicide?—Lay evidence will often settle the matter, but a medical examiner is expected to note all indications on the body and in the surroundings which may help to elucidate the question. While

he should note *everything*, the chief points to attend to are the position of body with reference to weapons and surrounding objects; evidence of a struggle from wounds on the hands, etc., and from surrounding objects; bloodstains on the body, on the surroundings, and on any weapon found; the possibility of the wounds having been produced by the weapon, the nature of objects (if any) grasped in the hands.

Bruise, contusion.—These terms denote an injury in which there is solution of continuity of the tissues and extravasation of blood without loss of external continuity. In superficial bruises discoloration due to effused blood appears at the site of injury in a short time; in deeper-seated bruises it will take longer to reach the surface. As the effused blood makes its way to the surface in the connective-tissue planes, the point at which the discoloration appears does not necessarily indicate the point at which the violence was applied—e.g. in the arm pit the discoloration appears below the part injured, and in the iliac and hypogastric regions above the part struck. As stated above, in a post-mortem examination all discolorations supposed to be due to violence should be incised to demonstrate the presence of effused blood.

If violence has produced fatal internal injuries, no bruises may be observed in making an external examination; thus, a blow on the abdomen may cause rupture of spleen, liver, intestine, or urinary bladder (especially when this is distended) without leaving any external mark of violence.

The amount of blood extravasated is not always in direct proportion to the force used, but varies in different persons and in different situations. Women and children are more easily bruised than those whose tissues are firmer. Very slight force will produce large extravasations of blood where the tissues are loose and vascular, e.g. in the vulva and scrotum. In scurvy, purpura, hæmophilia, and malignant cases of infectious disease, extravasations of blood occur spontaneously, and slight violence will produce extravasations out of all proportion to the force employed.

The date at which a bruise was inflicted can be judged only approximately from the colour—which, at first blue or bluish-black, becomes violet and successively green, yellow, and lemon before disappearing. The changes begin at the circumference.

Incised wounds are produced by sharp-edged

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instruments. The edges are usually regular and clean-cut, and the wound gapes more or less, owing to the tension of the skin drawing the edges apart. Irregularity of the edges may be met with when a blunt knife has been used or where the skin has been folded. The length of an incised wound gives no idea of the length of the cutting edge, as the point of the knife may have been drawn along and made to divide a considerable length of tissue.

Wounds made by a blunt instrument may simulate incised wounds when situated in soft tissues immediately over bone, e.g. the shin, the orbital ridge and other regions of the scalp, the line of the lower jaw, and the external genitals of females. The violence causes the soft tissues to split, but careful examination with a hand lens will usually show small bridges of tissue passing from side to side across the gap, and thus distinguish the wound from one made by a cutting instrument. In cut-throat wounds the issue between suicide and homicide cannot always be decided definitely from the wound, though the probability of its infliction in a particular way may be asserted. In non-fatal cases of suicide the wound is commonly in the upper part of the throat and, while opening the larynx, does not involve the large vessels. In fatal cases of suicide the whole of the soft tissues on both sides may be severed right down to the vertebral column. A suicidal wound may be tapering at the end from weakness caused by loss of blood at the first incision, but a homicidal wound may easily have the same characters. In suicidal wounds by a right-handed person the direction of the cut is from left to right transversely across or obliquely from above downwards. If a murderer cuts the front of the throat from behind, the direction of the wound will be the same. Wounds having a direction from below upwards are more likely to be homicidal, though an insane person may cut in any direction. Homicidal wounds often have the ends undercut, while in suicidal wounds the skin itself is the farthest point wounded. The terminal end of an incised wound of the throat is often serrated owing to the skin having been dragged forwards in folds by the weapon. This appearance, if present, will enable the examiner to decide if the wound was inflicted from left to right, or vice versa. Small tentative cuts are often found near the commencement of the main incision. If these are approximately parallel to the principal wound it will be in favour of suicide, but if in different directions

it will raise the presumption of homicide, the probability being that they were inflicted in a struggle to evade the assailant. The hands of the victim should always be examined. In a case of homicide, unless the victim has been taken by surprise or while asleep or unconscious, the hands are likely to be injured by grasping the weapon in attempts at self-defence.

Punctured wounds are produced by a weapon being driven straight in through the tissues. Their depth greatly exceeds the size of the surface wound. They may be produced by sharp-pointed weapons with or without a cutting edge, by comparatively blunt weapons such as a poker, or by pieces of broken glass or earthenware, etc. Where a sharp-pointed weapon such as a knife has been employed the surface wound is fusiform owing to greater retraction of the tissues in the middle of the wound. The length of the surface wound may exceed the breadth of the blade which produced it, from the wound having been enlarged during the withdrawal of the weapon. When a blunt-pointed instrument has been employed, the skin stretches before being perforated and retracts on withdrawal of the weapon, so that the hole is a little smaller than the diameter of the weapon. When a limb has been traversed the exit wound is usually smaller than the wound of entrance, as weapons capable of producing such wounds are usually tapered in shape. The edges of the entrance wound are sometimes everted by the withdrawal of the weapon. The tissues round a punctured wound may be contused when the weapon has been driven in up to the hilt, and this even when a sharp knife has been used.

The term *penetrating wound* should be reserved for a wound which enters a body cavity, e.g. the abdomen.

Lacerated wounds are produced by blunt instruments or by falls, and exhibit tearing as distinguished from cutting of tissues. They are often irregular in shape and have ragged edges, and bridges of tissue may be found passing from side to side across the gap. They are generally accompanied by more or less contusion of adjacent parts.

Contused wounds.—By a contused wound is meant a solution of surface continuity plus bruising of the surrounding tissues. Such wounds may be produced by a weapon, or by a fall, or by a blow of the fist, etc. It would be better to designate wounds as incised or lacerated, according as produced by sharp-

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edged or by blunt instruments, and to note any concomitant contusion.

Fractures.—Fractures of the skull are the most important; they may be due to direct violence from a blow or a fall, or to indirect violence from transmitted force, as when a person in falling lands on the feet or buttocks. When the fracture is due to direct violence applied with great force—e.g. by a pointed weapon or a hammer—a localized depressed fracture may result. In such cases the fracture of the outer table will correspond more or less closely in shape and size with the contact area of the weapon, whereas the inner table will be splintered over a wider area. When less force is used the fracture is usually diffuse and irregular. When the skull is free to move, fracture is found at the point at which the violence was applied, whereas when the head is fixed the fracture may start at the point of impact or at the opposite point—e.g. in falls on the vertex the fracture may begin either on the vertex or at the base. When the skull has been compressed from before backwards, as by the wheel of a heavy vehicle passing over the brow or occiput, it may fracture laterally. While depressed fractures may be expected to be associated with more or less extensive destruction of brain substance, even a simple fracture may lead to compression of the brain as the result of hæmorrhage from rupture of the middle meningeal artery or one of its branches. Injury to the brain is found not only at the seat of violence but sometimes on the opposite side, due to contrecoup. Contrecoup lesions always lie in the line of force of the impact, and may be more severe than those at the point where the violence was applied. The medical witness will usually be asked, "Was the fracture due to a blow or to a fall?" and "Was the hæmorrhage due to disease or violence?" The answers are by no means always easy, and the possibility of a fall having produced the fracture will frequently have to be admitted. Help in coming to a conclusion will be derived from the number and nature of the injuries, the thickness of the skull, the nature of the ground or object on which it is alleged the person fell, the manner in which he fell, and the position of the injury. In a simple fall the vertex is not likely to be injured unless in falling the top of the head comes into contact with a wall or other object. **Hæmorrhage** on the surface of the brain or between the dura and the bone is usually the result of violence, while hæmorrhage within

the brain substance is commonly due to disease. All the factors must be considered, and special attention directed to the state of the cerebral vessels, the condition of the kidneys (whether cirrhotic or not), the possibility of the deceased person having suffered from scurvy, purpura, or hæmophilia, and the presence or absence of meningeal disease. Pachymeningitis hæmorrhagica may cause extensive subdural hæmorrhage.

Concussion of the brain may accompany fracture, but also occurs from direct or indirect violence independent of fracture. As opposed to shock (in which there are no naked-eye changes) the condition is characterized post mortem by bruising or small lacerations of the brain tissue.

Fractures in the trunk or limbs may be caused by violence inflicted by another, or may result from falls. They also occur from muscular violence (e.g. fractured ribs, transverse fracture of the patella) and spontaneously. In fragilitas ossium and mollities ossium, owing to the increased fragility of the bones, fractures may be produced by very slight violence, or may occur spontaneously. Generally the bones of old people are more brittle and more easily broken than those of younger adults. Increased fragility of the bones is not infrequent among the insane, and the occurrence of fractures sometimes leads to charges of violence being made against attendants. In making an autopsy in fatal cases of this nature, trial should be made of the force necessary to produce fractures; it will sometimes be found that the ribs can be snapped with the fingers. In bodies taken from navigable waters, fractures and other injuries produced post mortem by the propeller of a ship, etc., are frequently found. The question of ante- or post-mortem production will depend on the presence of hæmorrhage and of signs of vital reaction.

In recent cases the age of a fracture may be estimated by the stage of repair after considering the age and the general health of the injured person. In the old and debilitated repair is often extremely slow.

Gunshot wounds.—Wounds produced by firearms vary in appearance according to the kind of weapon used, the distance at which it was discharged, the type of projectile, and the nature of the charge. It is impossible to lay down precise rules, as what applies to one firearm does not apply to others. Experiment should therefore be made, when possible, with the weapon used and with similar projectiles

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and charges. Usually the most important question to determine is the distance from the victim at which the missile was discharged.

A "near" wound has the following characters. There is a zone of blackening round the entrance wound, due to particles of unconsumed powder embedded in the skin and to the smoke of combustion. Any hairs round the part are singed, and the skin may appear parchment-like from the flame of combustion. The wound is often irregular in shape, even when a round bullet has been used, and may be much larger than the missile. This is due to the tearing up of the tissues by the gases of the explosion as well as by the bullet. The farther the weapon is held from the body the less marked will be these appearances.

With a modern revolver, even a near wound may have characters different from those described. The bullet is usually small and conical, and produces an entrance wound which, instead of being rounded or irregular, is a mere linear slit. There is less blackening from carbon particles and smoke, as the charge is in the form of a cartridge and the powder may be smokeless.

An exit wound, if present, will show none of the effects of the explosion, is as a rule smaller, and may sometimes, but not always, show everted edges.

In gunshot wounds of the skull the fractures produced are often out of all proportion to the size of the bullet. The entrance wound frequently shows a bevelling of the edge of the bone internally and a more extensive fracture of the inner table, whereas the exit wound, if present, shows more extensive fracture of the outer table.

In "far" wounds the size of the entrance wound will correspond closely to that of the bullet. There is usually some inversion of the edges, and scorching of the tissues and deposits of carbon are absent. If there is an exit wound it may be similar to the entrance wound, except that there is a tendency for the edges to be everted. When a bullet strikes a bone it may drill a clean hole or, becoming flattened, may splinter the bone and carry splinters along its track.

When a shot-gun has been employed the appearances vary greatly according to the distance from which the piece was fired. If it was fired close at hand the shot will have entered the body as one mass and caused a single entrance wound, but with great destruction of internal tissues from the spread of the

shot. If it was discharged at some distance there will be separate shot wounds. In these cases no opinion as to distance can be given until after experiment, and then only with reference to the charge and kind of powder and shot used.

In gunshot wounds the question of accident, suicide, or homicide is not always easy to determine. Self-inflicted (accidental or suicidal) wounds are necessarily near wounds. Self-inflicted accidental wounds may be found in practically any situation, even in the back, their occurrence in unusual situations being generally due to carelessness, as in climbing over a fence with a loaded weapon at full cock. In suicidal cases the weapon is occasionally found firmly grasped in the hand as a result of instantaneous rigor, but more frequently is found lying close to the body. As a rule, suicidal wounds are directed to a vital part, e.g. the heart or brain. A right-handed person will most commonly reach his brain by firing through the right temple, or the muzzle of the weapon may be placed within the mouth, which is almost impossible in the case of murder.

The direction of the wound is of primary importance, as, even when the entrance wound is in a position accessible to the deceased person, the direction may be inconsistent with self-infliction. The possibility of deflection occurring after the missile has entered the body must, however, always be remembered.

Homicidal wounds may be situated anywhere, and as a rule these are not near wounds. A far wound will always raise the suspicion of homicide. The position of the wound may favour homicide, but what has already been said with regard to accidental wounds should be borne in mind.

When there is no wound of exit the missile should be carefully looked for in the tissues, and preserved when found. Even if there is a wound of exit, it is possible that portions of the projectile may be found in the body.

The immediate causes of death in gunshot wounds are injury to vital organs, shock, or hæmorrhage; later, death may result from secondary hæmorrhage or sepsis.

A. ALLISON.

INJURIES, HEAD (see **HEAD INJURIES**).

INJURIES, SPINAL (see **SPINAL INJURIES**).

INJURIES, TREATMENT OF (see **WOUNDS, TREATMENT OF**).

INSANITY, CERTIFICATION OF

INSANITY, ADOLESCENT (*see* DEMENTIA PRÆCOX).

INSANITY, CERTIFICATION OF.—In the Middle Ages persons of unsound mind—chiefly because mental disorder seems to have been ignored or misinterpreted until the conduct of the patient eventuated in outrage—were not always differentiated from criminals and were, with them, housed in prisons. As time went on, the desirability of separating insane persons from criminals became manifest, though the institutions in which the former were interned differed but little in appearance and management from the prison. To such places were consigned the undoubtedly insane and at times, as it was alleged, sane persons who were inconvenient to their friends and relatives. The popular imagination was chiefly inflamed as to the unlawful detention of sane persons by the publication of certain novels which in their day created no small stir. The result of the agitation can be seen in the Lunacy Acts, the framers of which were concerned more for the rare individual who might possibly be detained improperly and who was mostly a creature of the imagination, than for the mental patient. The Acts make the treatment of patients who are mentally disordered practically impossible unless they are certified—which means, in a vast majority of cases, unless they are sent to asylums. For this reason, and because of the social disabilities attending a reputation for unsoundness of mind, certification, rightly or wrongly, is objected to by most patients and by such friends and relatives of patients as are genuinely concerned for their welfare, and is postponed until the last moment. The result is that treatment also is postponed; or, if the doctor is called in, treatment is carried on at a medical disadvantage and at legal risk to those concerned in it, the affection passing as “nervous” or as anything rather than itself.

There are, however, various reasons which in many cases render recourse to asylum treatment necessary. In the forefront is the question of expense. It is obvious that if a patient is to be housed away from home—and this is desirable in many cases, if he is to be adequately nursed and to be medically visited once or twice a day—the monetary outlay will be considerable, and in a very large majority of cases utterly prohibitive. Medical and nursing care in an institution is then the only possible course, and, unfortunately, admission

to an institution can only be obtained when the patient is duly certified and when the institution is recognized by licence or otherwise. That such a person cannot, when there is no plain medical reason against it, be admitted to the ordinary wards of a general hospital, or to special wards in connexion with a general hospital, is a very great disadvantage both for patients and for medical men, but that at present is unhappily the case. In some instances violence, noisiness, or the nature of the delusions necessitates physical restraint, or refusal of food makes forcible feeding imperative, and in these cases legal recognition of the necessity for such restraint and treatment is indicated.

The character of the asylum population indicates that the principal use of such institutions is to afford shelter for the incurable. Here again the question of expense governs the situation. The combination of cheapness and efficiency is in most cases found at a maximum in an institution, although unfortunately the patient is usually lost in the crowd of his fellow-unfortunates and gets but little individual attention. There is no doubt that the level of many of the demented could be raised very considerably if patients received some measure of re-education, but it is obvious that directly personal effort and time are given, expense is increased. Considerations such as these must influence a decision as to whether a patient for whom there can be small hope of recovery should be placed in an asylum or in private care, or kept at home.

A patient of unsound mind can be sent to and detained in an institution or in private care in various ways. The following is the method most usually adopted: (1) *Medical certificates* are made out by two registered practitioners, and must be signed not more than seven clear days previously to the date of the presentation of the petition. Where feasible, one of these should be the patient's usual medical attendant. The practitioners signing the certificates must each examine the patient separately and apart from each other. The following cannot sign certificates, viz. the petitioner; if the order is an urgency order, the person signing it; the superintendent, proprietor, or medical attendant of the asylum, hospital, or house to which it is intended that the patient should go; any person interested in the payment or accounts of the lunatic; the husband or the wife, father or father-in-law, mother or mother-in-law, son or son-in-

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law, daughter or daughter-in-law, brother or brother-in-law, sister or sister-in-law; the partner or assistant of any of the foregoing persons. The practitioners signing the certificates must not be in partnership, nor one the assistant of the other; nor must they be related to one another, as father, father-in-law, mother, mother-in-law, son, son-in-law, daughter, daughter-in-law, brother, brother-in-law, sister, sister-in-law. (2) A *petition* has to be made requesting the judicial authority—that is, a stipendiary magistrate, a county court judge, or a justice appointed for the purpose—to make an order for the reception of the patient. The petitioner must, where possible, be a relative of the patient, and must have seen him within fourteen days of the presentation of the petition. The petition must be presented not more than seven clear days after the date of the medical examinations. It is accompanied by (3) a *statement* of particulars concerning the patient, such as his age, status, religion, personal and family histories. The petition, statement, and medical certificates are laid before the judicial authority, who thereupon makes (4) an *order* for the reception of the patient in the institution or house selected. It is not necessary for the judicial authority to see the patient, but he may if he so desires. When the order has been made the papers are complete, and the patient must be received within seven clear days. If he is not received within that time the papers become useless and the whole business has to be gone through again.

In the event of its being imperative or desirable that the patient should be brought at once under care, an *urgency order* may be made. The order is made by a relative or friend and is accompanied by only one medical certificate. There is no petition to, and order from, a judicial authority. The patient must have been seen by the relative or friend making the order within two days of its being made, and must be admitted to the institution or home selected within two days from the date of the medical examination. A patient may be detained for seven days from the date of the order, and within that period the ordinary papers must be filled in. In the case of pauper patients the best course is for the relatives or friends to inform the relieving officer of the district. This official acts according to the prescriptions as to summary receptions which are described below.

Printed forms for certificates, petition, state-

ment, and order can be obtained of law stationers. To avoid trouble with the Commissioners, the minute directions which are printed thereon should be strictly carried out. Only facts indicating insanity should be communicated. Abnormal physical signs, or clinical pathological findings, and the medical man's deductions from the facts or his diagnosis, are of no interest and only lumber up the certificate.

Patients can be brought under care by *summary reception orders* in the circumstances now to be detailed.

1. It is the duty of a constable, relieving officer, or overseer, if it comes to his knowledge that a person, not a pauper and not wandering at large, is not under proper care and control or is being neglected, to give information under oath to a justice. The justice authorizes two medical practitioners to examine the patient, and certify if necessary. On certification the justice makes an order that the person shall be placed under care.

2. It is the duty of the medical officer of a union, who has knowledge that a pauper resident within his district is a lunatic, to give notice to the relieving officer or overseer, and of such officer or overseer thereupon to give notice to a justice, who orders the relieving officer or overseer to bring the lunatic before him.

3. It is the duty of every constable, or relieving officer, or overseer, who has knowledge that any person (pauper or not) wandering at large is a lunatic, to take such person before a justice. Or a justice upon information may order a constable, relieving officer, or overseer to bring an alleged lunatic before him. The justice then calls upon a medical man to certify, and orders the reception of the lunatic in an institution.

4. If a constable, relieving officer, or overseer is satisfied that it is for the public safety or for the welfare of an alleged lunatic, it is his duty to remove the patient to the workhouse of the union. The master of the workhouse may detain the alleged lunatic for not more than three days, and before the expiration of that time the constable, relieving officer, or overseer must take proceedings as described in preceding paragraphs.

5. If a justice is satisfied, in any case where a summary order might be made, that it is expedient that a lunatic should forthwith be placed under care, he may make an order for taking the lunatic to a workhouse; an order

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so made does not authorize detention for more than fourteen days.

Another way by which a patient may be brought under care is by order of the Board of Control. Two or more of its members may visit a lunatic not in an institution or workhouse and, if they think fit, call in a medical practitioner to examine the patient. If the practitioner certifies, the members of the Board of Control may order the lunatic to be received in an institution.

In the case of persons of property the extensive procedure of an inquisition in lunacy may be held before a Master in Lunacy, or a Judge of the High Court and a jury, to decide upon the sanity of a patient and his fitness to look after himself and his property. A committee of the patient's person and a committee of his estate may be appointed respectively to look after the one and the other.

E. D. MACNAMARA.

INSANITY, CIRCULAR (*see* FOLIE CIRCULAIRE AND RECURRENT INSANITY).

INSANITY, CONFUSIONAL (*see* CONFUSIONAL INSANITY).

INSANITY, DELUSIONAL (*see* DELUSIONS; PARANOIA).

INSANITY, EPILEPTIC (*see* EPILEPTIC INSANITY).

INSANITY, ETIOLOGY OF.—Any consideration of the etiology of insanity must be prefaced by three statements. In the first place, insanity is not a medical or scientific entity, but simply a legal term indicating a degree of mental disorder which renders the patient unfit to control himself and his affairs, and which makes it necessary for him to be placed under care and restraint. A patient with a particular form of mental disorder may or may not be insane, according to the extent to which the disorder influences his conduct. Whether the case falls on one or the other side of this legal line has clearly no significance from the etiological point of view, and the discussion which follows will therefore deal with the origin of mental disorder as such, in whatever degree it may exist. In the second place, mental disorder is an extremely wide conception and includes within its boundaries a great diversity of distinct conditions. Hence it is as unsatisfactory to group together in a single article the etiology of all these distinct conditions as it would be to attempt a similar

task in the case of physical disorder. Lastly, our knowledge of the causation of mental diseases is at present very imperfect, and in only a few conditions has a definite chain of cause and effect been established. So far as the greater part of the field is concerned, we can only say that in cases of mental disease certain antecedent factors are found to exist with a frequency which suggests that a causal relationship is present. Of the causation of insanity in the sense of an exact and predictable sequence between cause and effect—the sense in which it would be understood, for instance, by a bacteriologist—we know very little. As a consequence, the nature and significance of the factors which precede mental disorder is a matter largely of surmise and opinion, and hence of dispute.

In the light of these considerations we shall here only attempt to enumerate the antecedent factors whose causal relationship to mental disease is presumed on the grounds stated above, with a clear understanding that our knowledge of the precise mode of action of these factors is very inadequate, and that no exact information as to their relative importance can be given.

A distinction is generally made between *endogenous* and *exogenous* factors—that is to say, between the causes which are regarded as inherent in the constitution of the individual, and those which are due to interaction between the individual and his environment. The former group is practically comprised in the conception of heredity, the latter are very numerous and may be further subdivided into two main sub-groups, physical and psychical.

Heredity.—It is beyond question that heredity is an important, and possibly it is the most important, factor in the causation of insanity. If the family histories of a number of insane patients are compared with those of a similar number of sane patients, it will be found that the percentage of insane and neurotic ancestors and collaterals in the former is very much greater than in the latter. It is necessary to realize, however, that that is almost all that our present knowledge permits us to say with certainty. We cannot predict whether the offspring of any given union will be insane; we find many normal persons in families with a heavy hereditary taint, and we find insane persons in families in which no such taint can be discovered.

The chief facts at present at our disposal with regard to the incidence and mode of action

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of heredity may be summarized as follows: Insanity is not directly transmitted as such, but merely as a tendency; that is to say, the individual possesses a potentiality which may or may not develop into an insanity, according to the stresses to which he is subjected throughout his life. Moreover, the factor transmitted is not the tendency to develop the particular type of insanity exhibited by the ancestor, but a more general factor which may develop into one or other of several distinct conditions, some of which do not belong to the insanities at all. For example, the descendants of an insane parent may exhibit one of many different types of insanity, mental deficiency, epilepsy, hysteria or other neurosis, or alcoholism. Similarly, in the ancestry of an insane patient any or all of these conditions may be found. The facts just described are regarded as due to the operation of "dissimilar heredity," and the factor transmitted is held to be a "neuropathic constitution," which is either a favouring or necessary condition of the growth of one or other of the diseases mentioned above.

Although heredity probably plays some part in all forms of insanity, its incidence is very much greater in some than in others. In general paralysis, senile dementia, and the toxic psychoses, for example, the taint is often slight or apparently absent; in manic-depressive insanity and in dementia præcox it is considerably greater; while in imbecility and idiocy it is generally very pronounced. There seems to be good evidence for the statement that mental disorder tends to appear at an earlier age in each succeeding generation, so that the children of a parent who became insane in middle age are more likely to develop some form of insanity in adolescence. This statement has been termed the "law of anticipation."

It is often said that the marriage of cousins or other form of inbreeding is likely to lead to insanity in the offspring. There appears, however, to be no justification for this generalization. If the parents belong to an untainted stock the children should be equally untainted. If, on the other hand, the stock is tainted, then the chances of insanity occurring in the offspring of a consanguineous marriage are naturally greatly increased. This is probably the explanation of the prevalence of insanity in certain races and stocks where inbreeding is common.

In recent years attempts have been made to bring the inheritance of mental disorder

under Mendelian laws. The extensive researches of Rossanoff and Orr have elicited facts which certainly support such an interpretation, and if these results are ultimately established our knowledge of the incidence of heredity will become far more precise, and many of the preceding statements will require considerable modification.

The chief problem concerning heredity with which the practitioner is likely to be faced is the giving of advice with regard to the marriage of one whose family is tainted with insanity or other evidence of the neuropathic constitution. His decision must take into account all the facts detailed above. To begin with, it must clearly be understood that, owing to the limitations of our knowledge, it is very difficult to make any dogmatic statement, and only probable risks can be inferred. The grounds upon which the principal stress must be laid are the mental and physical state of the person concerned, the proportion of normal and of neuropathic members in the ancestry, and the type of disorder which the latter have exhibited. Where, for example, the individual is healthy, the proportion of neuropathic ancestors very small, and the types exhibited are disorders such as general paralysis or senile dementia, the risk is comparatively slight. If both the proposed parties belong to tainted families the risk is clearly grave.

Exogenous etiological factors.—The relative importance of the mental and physical groups is a subject of much dispute, and probably the most satisfactory statement that can be made at the present time is that both may furnish etiological factors, that their relative weight differs greatly in different forms of mental disorder, and that the actual development of insanity is likely to be brought about by a concatenation of causes belonging to both groups.

The *mental factors* are emotional shocks and stresses of all kinds, worries, anxieties, and so forth, practically all of which can be summed up in the modern conception of intrapsychic conflict. Certain schools of thought claim to have demonstrated that these conflicts, when analysed into their ultimate constituents, are due to errors of development in the great primary instinctive forces, particularly that of sex, but these conclusions are not as yet generally accepted.

The *physical factors* which may play a part in the etiology of insanity would seem to be

legion, and it would probably be correct to say that almost any disturbance of the bodily economy may act as a contributory cause in the production of mental disorder. With few exceptions, however, no definite relationship between the alleged cause and the resultant disorder can be established, and the attempt to erect such entities as phthisical insanity, puerperal insanity, and so forth, has no adequate foundation. The chief exceptions referred to are syphilis and alcohol. Syphilis is the direct cause of general paralysis, and alcohol produces a fairly definite series of mental disorders—delirium tremens, alcoholic hallucinosis, and various chronic alcoholic conditions. Gross injuries do not appear to have any precise causal relationship to insanity, although certain cases of mental deficiency are directly due to this cause. The dementias, however—senile, arterio-sclerotic, alcoholic—are ascribable to definite generalized lesions in the brain.

Acute infections are occasionally followed or accompanied by mental disorders which have a fairly characteristic symptomatology, and which form a well-marked clinical group—deliria, confusional psychoses, Meynert's amentia. Although these disorders also occur after severe emotional and physical stresses, they are generally regarded as toxic in origin. Insanity occurring during the puerperium is often of this type.

The part played by the endocrine glands in the causation of insanity is at present very uncertain, although forms of mental deficiency (cretinism) are unquestionably to be ascribed to affections of this system. It seems clear, however, that a body of evidence is now accumulating which ultimately will cast a great light on the etiology of insanity, and that in the disturbances of the endocrine glands and their associated systems will be found the solution of many obscure problems. It is likely, moreover, that in this direction we may look for a common ground upon which the conflicting schools that ascribe the causation of mental disorder predominantly to psychical factors on the one hand, and to physical factors on the other, may reconcile their claims.

Finally, a word may be said with regard to an error very commonly made in estimating the significance of alleged etiological factors—the error of confusing causes with symptoms. One of the most striking instances is to be found in the part often ascribed to alcohol. Alcohol may certainly be a cause of insanity,

but alcoholic and other excesses may be merely a symptom of an insanity already established, and in the causation of which alcohol has had no place. Similarly, financial and other anxieties are in some cases merely symptomatic, and due to the fact that the patient is regarding in a distorted light a financial or other situation which in itself provides no ground for apprehension.

BERNARD HART.

INSANITY, INTERMITTENT (*see* FOLIE CIRCULAIRE AND RECURRENT INSANITY).

INSANITY, MANIC-DEPRESSIVE (*see* MANIC-DEPRESSIVE INSANITY).

INSANITY OF EXHAUSTION (*see* CONFUSIONAL INSANITY).

INSANITY, PUERPERAL (*see* PUERPERAL INSANITIES).

INSANITY, RECURRENT (*see* FOLIE CIRCULAIRE AND RECURRENT INSANITY).

INSANITY, TREATMENT OF.—This subject may be considered under the headings of Prevention and Curative Treatment.

1. **Prevention.**—The scope of preventive measures is too wide to be dealt with fully within the limits of this article; it includes questions of marriage, social reform, and education.

Marriage between near relatives when there is a strong family history of insanity or other nervous disease (e.g. epilepsy) must always be fraught with danger. The crying scandal of weak-minded women bearing numbers of illegitimate children, who eventually become a burden on the rates, is to some extent diminished by the Mental Deficiency Act, and will be more so in the future. The public now take a great interest in this matter, and physicians must be prepared to give intelligent counsel.

Social reform is a big subject, of which only the main bearings can here be touched upon. Syphilis, the abuse of alcohol, and addiction to drugs such as morphia and cocaine, are undoubtedly the causes of many cases of insanity. The measures which are being taken to cope with these evils are well known. The proper education of children of neurotic parents is a matter of great importance, and the family physician should discourage hard study and everything in the nature of "cramming" and attempts at prize-winning. Caution is especially necessary as the child approaches the age of puberty. The importance of a sufficiency of sleep is now better understood than it

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used to be ; in "nervous" children, especially, early hours are essential. This warning is all the more necessary now that "summer-time" regulations tempt all to later hours.

Stress must be laid upon the choice of a suitable avocation. In the case of an unstable youth or girl no better advice can be given than "Back to the land." The changes produced by the War have provided facilities for an agricultural career for both sexes.

When an attack is threatened, much can be done by early treatment to avert it. The adoption of suitable diet, the correction of constipation, and the avoidance of exciting employment or amusement, with change of scene, may ward off an attack. The treatment of sleeplessness, which is usually an early symptom, will be easy before insomnia has become a habit. There is a growing agitation on the part of mental specialists to secure early treatment of insanity, which at present, owing to the Lunacy Acts, is difficult, and in some cases impossible, to obtain.

2. Curative treatment. (1) **General measures.**—The first question to arise in the mind of the practitioner called upon to deal with a case of mental disorder is, whether the case is suitable for home treatment or for an institution. In the acute psychoses, when fully developed, there is no difficulty in coming to a proper decision, but the early and border-line cases often cause perplexity. Here the question of diagnosis is most important. Should physical signs of general paralysis be present, it is essential that the patient should at once be placed under care, as by his extravagant schemes he may ruin himself and his family in a few days. The possibility of suicide must ever be borne in mind ; a tendency in this direction is difficult to guard against in the home. Finance also must play an obvious part in determining the question. Home treatment is, of course, expensive owing to the necessity for engaging the services of one or more nurses. If the patient is not to be kept at home, the law requires that he be certified (*see* INSANITY, CERTIFICATION OF). If the nursing is to be done in a private house, a room on the ground floor is essential, the windows should be blocked so that it is impossible to open them more than 5 in. at the top and bottom, and all dangerous articles such as knives, razors, scissors, fire-irons, etc., must be removed. If a patient, though not certifiable, is nevertheless desirous of having institutional treatment, he can enter a registered hospital or a private asylum as a

voluntary boarder after a few simple formalities have been complied with.

Though the mental symptoms in the acute insanities are apt to divert attention from the less prominent bodily condition, it must be remembered that the sufferer is really ill and requires rest in bed, if possible in the open air. Most institutions are provided with verandas which can be occupied by night as well as by day, and so appetite is stimulated, excitement diminished, and sleep promoted. Prolonged hot baths at a temperature of 96° F. are valuable in cases of excitement, whether maniacal or otherwise. It is best to commence with half an hour for the first day, and gradually to increase the time spent in the bath as the patient can bear it. Three or four hours is the maximum time in most cases. The cold shower bath, or cold bath on rising, is often appreciated as a general tonic. Massage and movements of the joints should be used in cases of stupor, to prevent deformities. Electrical treatment is given in some institutions, and one of the best methods of applying it is in the form of the sinusoidal current in a bath. The faradic current is sometimes resorted to in cases of stupor, but with little effect. The Turkish bath has been said to give good results in certain cases.

Foreign travel used to be largely prescribed, especially in cases of melancholia. The reason given was that an endeavour should be made to "rouse" the patient. This is, of course, erroneous, for it is rest that is required, and the risks, especially in connexion with suicide, are increased tenfold. Travel is useful, however, when the patient is convalescing, and before he returns to his former life a visit either to the Continent or to the seaside should be recommended.

In the rare event of extreme excitement causing a patient to be uncontrollable by the nursing staff, the question will arise as to the advisability of tying him down in some way. This is known as "mechanical restraint." Many were the atrocities committed in former years by means of chains, handcuffs, etc., and the pioneers of the non-restraint system had to encounter numerous adversaries and grapple with difficulties. The obstacles were gradually overcome, and now in many institutions no means of mechanical restraint exist. The practitioner, if he has charge of a patient, must remember that only certain forms of mechanical restraint are permitted, and that, if they are used, the reason, and the means employed,

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must be entered in a book kept for the purpose. It must be emphasized that such appliances should be resorted to only when everything else fails. The word "seclusion" means locking a patient in a room by himself between the hours of 7 A.M. and 7 P.M. This also has to be recorded, and, like mechanical restraint, must be kept at the lowest possible limit.

When the acute symptoms have passed away, carefully regulated exercise should be ordered, while in chronic cases the value of work must not be forgotten. Every effort must be made to prevent the patient from sinking into a degraded state. Even in dementia a good nurse often succeeds in maintaining in her charge habits of cleanliness and order. In the congenitally defective, wonderful results have been obtained by trained instructors in various crafts. Amusements, sports, religious services, etc., find a valuable place in the scheme of treatment in all institutions.

(2) *Diet*.—In the acute insanities the coated tongue, the foul breath, and the constipation that are present are sufficient evidences of disordered alimentation. At first it may be necessary to give diluted milk for a few days. This may soon be supplemented by eggs beaten up in the milk and by the easily digested sugars found in invalid foods. The normal manner of living must be resumed gradually, as in other diseases. In melancholia, when the digestive organs are working satisfactorily, much good may be obtained from overfeeding with milk and eggs and with fats such as cream and cod-liver oil—Clouston's "gospel of fatness"—and every means should be taken to bring about increase in weight. Epilepsy is often associated with insanity, and in such cases a diet with a minimal amount of animal protein is indicated. In general paralysis of the insane the food must be minced owing to the voracious appetite and the paresis of the muscles of deglutition met with in these cases. With regard to alcohol, as a general rule it is to be avoided as far as possible, especially, of course, in cases in which it has seemed to play the part of a causative agent. In exceptional cases a little ale or stout may be allowed as an appetizer, and alcohol is of course to be given when required as part of the recognized treatment of any physical ailment occurring in the insane. Thus it should be given unsparingly in the exhaustion which follows acute excitement when the condition of the patient resembles that known as the "typhoid state."

(3) *Medicinal treatment*.—The treatment of

any given case is largely made up of treating the symptoms as they arise. When the cause is known and removed, as in alcoholic insanity, recovery quickly follows; but unfortunately the cause is unknown in most cases of insanity. The special treatment required by the different varieties of insanity, e.g. melancholia, will be found described in the articles on those subjects, and only the general therapeutic outlines will be considered here.

(a) *Constipation*.—In the majority of cases this is present, and is apt to be neglected, as information from the patient is unreliable or unobtainable. It is, however, of primary importance that the bowels should act freely. Doses larger than in ordinary practice may be required. No special aperient is indicated, but in obstinate cases the use of a teaspoonful of castor oil every hour till it acts, or one-sixth of a grain of calomel employed similarly, proves effective. It is not uncommon to prescribe as a draught 1 oz. of castor oil with one drop of croton oil, the whole made into an emulsion with liquor potassæ 15 min. and water up to 2 oz. If the above measures are unavailing, enemata of various kinds may be tried; occasionally it is necessary to clear out the rectum with the finger.

(b) *Excitement*.—The use of sedative drugs should be limited as far as possible. On the other hand, long-continued and severe excitement weakens the heart and generally exhausts the patient, and the lesser of the two evils has to be chosen. Hyoscine hydrobromide given hypodermically in doses of $\frac{1}{100}$ gr. to $\frac{1}{50}$ gr. is, as a rule, reliable. The large class of drugs known as hypnotics all have a sedative action. It will be found in practice that reliance will be placed to a great extent upon sulphonal, chloral, and the bromides. There are several dangers associated with the use of sulphonal for long periods. As it is slowly absorbed and may accumulate in the intestines, it is best given as an emulsion; the bowels should be kept acting well and the administration intermittent. It sometimes causes hæmatoporphyria; that is to say, a pigment called hæmatoporphyrin colours the urine, giving it an appearance like porter. Any sign of the urine becoming darker in a patient taking sulphonal should be viewed with suspicion and the drug stopped at once, otherwise death may result. Except in melancholia, opium is of little value in excited states.

(c) *Insomnia*.—Sleeplessness is often the first symptom to appear, and is one of the most

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distressing features of the illness. Before resorting to drugs, it is well to bear in mind that with the free action of the bowels and reduction of the general blood-pressure by means of simple remedies such as pil. hydrarg. followed by a saline draught, sleeplessness often disappears. In mild cases a glass of hot milk, or better still, if the patient is unaccustomed to alcohol, a glass of hot whisky-and-water, will break the bad habit upon which the sleeplessness may depend. If drugs are necessary it is well to try a dose of the mixed bromides at bedtime. If this is unavailing, paraldehyde in 1- or 2-drachm doses is a safe and usually a certain hypnotic. It should be given well diluted; with tincture of senega it forms an emulsion with water. It suffers from the serious drawback that it gives rise to a disagreeable odour in the breath the day following its use. Amylene hydrate belongs to the same chemical class and is without that disadvantage, but is probably less reliable in its action. Chloral often succeeds when other drugs fail. It is a cardiac depressant and is therefore feared, but in practice ill results are rarely seen, though of course in cases of heart disease it should be avoided. Veronal has made a position for itself, and in doses of 5 gr. is often effective. Luminal is a similar drug which at times acts powerfully in doses of 5 gr. Dial (the trade name for diallyl barbituric acid) has proved a safe and satisfactory hypnotic in doses of 1½ gr. taken at night. When sleeplessness is due to pain, opium is the only drug of value. This list could be prolonged indefinitely, but in the greater number of cases one or other of the drugs mentioned will be all that is required. The combination of two or more hypnotics is often successful when one fails. A useful plan is to give a dose of sulphonal at teatime and then a dose of paraldehyde or of veronal at bedtime.

(d) *Refusal of food.*—This is frequently either the result of a delusion or the means adopted to attempt suicide, so that argument will be unavailing and compulsion required. In forcible feeding the first essential is that the patient must be securely held. If there is much struggling, control will be most easily obtained by laying him on a bed. Three assistants will be required. A sheet is stretched firmly across the fully extended knees, which must be close together. The assistant on the patient's right will place his right knee on the sheet as close to the patient's thigh just above the level of the knee-cap as is possible with-

out pinching him. This assistant's right hand will be on the patient's right shoulder, and the assistant's left hand on the patient's right wrist. The assistant on the left of the subject will adopt a similar attitude, and the third will control the movements of the head. When the patient is under firm control he may swallow when a little milk is poured into his mouth, and this may be assisted by closing the nostrils. This should not be done in a feeble subject, as there is some danger of inhalation. If he spits the food out, the tube will have to be used—either a narrow one passed through the nose or a wider one through the mouth. The disadvantages of nasal feeding are the danger of passing the tube into the larynx and the fact that the nose becomes sore after a time. The disadvantage of mouth-feeding is the necessity of forcibly opening the mouth with a gag, but on the whole it is to be preferred, as a tube can be used which is too large to pass through the larynx. The food may be composed of milk and eggs; malted farinaceous foods, too, can be given in this way, and the nutritive value of cane sugar should be borne in mind.

The insane are not less liable than the sane to attacks of disease, and the treatment is the same in both classes. Epilepsy is frequently met with in asylums, and is then treated on ordinary lines, though in some cases of mental disease the sufferers seem to be generally better when having fits than when, under the influence of bromide treatment, the fits cease.

During convalescence tonics such as strychnine, arsenic, and iron will be required, and the patient must gradually be urged to interest himself in his surroundings and take up some form of occupation.

(4) *Psycho-therapeutics.*—The whole atmosphere of a mental hospital, the influence of the doctors, nurses, and attendants, often leads to cure by suggestion, perhaps not intentionally given. Further, it is impossible to exclude, as in other maladies, the beneficial influence played by suggestion in the remedies whose use is recorded under preceding headings. Apart from this, the measures commonly called psycho-therapeutic are rarely used in British mental practice, at any rate in the case of the definitely insane. Nor has psycho-analysis, in the manner recommended by Freud, Jung, and their followers, been much used in the treatment of the acute psychoses. In mild and border-line cases there is no doubt that much good often comes from prolonged

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conversations with the patient. When the various emotional factors which have played a part in the onset of the disease have been discussed, misconceptions removed, and a faulty environment corrected, some symptoms disappear, and others, such as hallucinations, become less persistent.

The use of hypnotism has not been found of much value in insanity, and though a few writers, such as Voisin, have recorded wonderful successes, their results have not been generally confirmed. Hypnotism, however, is occasionally effective in producing natural sleep when medicines have proved useless, though as a rule drug treatment is more certain. Hypnotism has had considerable success in reclaiming drunkards, and in alcoholic insanity it might be used to prevent relapses.

R. H. STEEN.

INSOLATION (*see* SUNSTROKE).

INSOMNIA (*see* SLEEP, DISTURBANCES OF).

INSULAR SCLEROSIS (*see* DISSEMINATED SCLEROSIS).

INTERMITTENT CLAUDICATION (*see* ARTERIAL DEGENERATION; ANEURYSM).

INTERMITTENT HYDRARTHROSIS (*see* HYDRARTHROSIS, INTERMITTENT).

INTERNAL CAPSULE, LESIONS OF (*see* NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF).

INTERSTITIAL NEPHRITIS, CHRONIC (*see* NEPHRITIS).

INTERTRIGO (*see* IMPETIGO CONTAGIOSA; ECZEMA).

INTESTINAL COLIC (*see* COLIC).

INTESTINAL INFANTILISM (*see* MORBUS CELLACUS).

INTESTINAL OBSTRUCTION.—A condition in which there is some obstacle to the normal progress of the contents of the intestine.

Etiology.—The obstruction may be due either to mechanical hindrances or to paralysis of the normal movements of the bowel induced by inflammation or toxæmia. Here we shall confine our attention to the mechanical forms of obstruction; the remainder are considered in connexion with the diseases by which they are caused.

The lumen of the gut may be occluded (*a*) by pressure or traction from without, (*b*) by

congenital stricture, or by new formations in the gut-wall, (*c*) by foreign bodies within the canal, or (*d*) by twisting or invagination of the intestines.

(*a*) **Pressure or traction.**—Pressure on the gut may be caused by any abdominal tumour, especially pelvic tumours. Large abscesses may also cause obstruction in the same manner. A much more common illustration of obstruction by external pressure is that of *strangulated hernia*.

Strangulated *external* hernia is easily the most common cause of intestinal obstruction. A portion of gut, by some sudden increase of intra-abdominal pressure, is forced through a weak spot in the abdominal wall (inguinal, femoral, umbilical, or ventral), and the margins of the opening constrict the gut-lumen while the vessels supplying the gut are also gradually occluded.

Strangulated *internal* hernia is comparatively rare, but the mechanism of obstruction is similar. The fossæ into which the gut may be forced are the duodeno-jejunal fossæ, the retrocæcal peritoneal recesses, the lesser sac of peritoneum (through the foramen of Winslow), the fossa at the base of the mesosigmoid, the recess of the obturator canal, and occasionally other and less recognized fossæ.

Peritoneal *adhesion-bands* frequently cause obstruction by pressure inducing constriction of a coil of gut.

Traction due to peritoneal adhesions does not cause quite an acute form of obstruction. Adhesions limit the normal movements of the gut and ultimately may produce a serious narrowing of the lumen. The adhesions may be congenital, but more commonly are the result of former inflammatory disease, e.g. appendicitis, salpingitis, or peritonitis.

(*b*) **New formations in the gut-wall** may be either tumours, malignant or benign, or inflammatory products.

Cancer is a common cause of obstruction of the large bowel, while tuberculous inflammatory products may cause obstruction at the ileo-cæcal junction or in the small intestine (*see* INTESTINE, NEW GROWTHS OF; and INTESTINE, TUBERCULOSIS OF). The fibrous stricture which forms as the result of previous ulceration of the small intestine should be included under this heading.

(*c*) **Foreign bodies** within the canal rarely cause obstruction. A gall-stone is the most common substance foreign to the intestine which may cause occlusion, but occasionally

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swallowed metal or similar objects, or collections of indigestible food residue, may bring about the same result.

(d) **Twisting or invagination of the intestines.**

—A twisting of a coil of intestine on its own mesenteric axis or around another coil of gut is termed a *volvulus* (q.v.) An *invagination* of one portion of gut into the part immediately below it is termed an *intussusception* (q.v.).

Age-incidence of intestinal obstruction.—

Under 2 years of age the commonest form of intestinal obstruction is *intussusception*. Strangulated external hernia is responsible for more than half the total number of cases in adults. Apart from hernia, cancer of the large bowel is the most frequent cause of intestinal obstruction in old people, whilst in youth and middle age strangulation by bands, obstruction by adhesions and internal hernia comprise the main causes.

Pathology.—The pathological changes vary according to the acuteness and the completeness of the obstruction.

In *acute cases of complete obstruction* the intestine above the obstruction undergoes violent peristaltic contraction to no avail, and the intestinal contents are dammed back. Gas accumulates and causes increasing distension, and clear fluid is effused into the peritoneal cavity. The gut below the obstruction remains contracted and empty. When a coil is strangled the pressure on the blood-vessels (first the veins, then the arteries) causes asphyxia and devitalization of the gut-wall with ensuing gangrene. This change takes place slowly or quickly according to the degree of the vascular obstruction.

In *chronic, subacute or incomplete obstruction* the increased force requisite to propel the intestinal contents causes hypertrophy and distension of the gut above the obstruction.

If a case of obstruction be neglected the accumulation of decomposing material in the distended coils of gut causes severe toxæmia, and in some cases the diseased intestine allows organisms to enter the peritoneal cavity and thus leads to peritonitis.

When any considerable portion of mesentery is strangled the pathological phenomena of shock are strikingly evident, and the same holds good for any acute obstruction of gut without mesenteric involvement.

Symptomatology.—*Pain* is the most constant of all the symptoms. In acute obstruction it may be agonizing; in subacute and chronic cases it is proportionately less. It

tends to come in spasms corresponding to the peristaltic waves of the intestine, but in strangulations is more continuous. In chronic cases the patient and the doctor may mistake the colicky pain for indigestion unless a careful examination be made. The site of the pain is usually epigastric or umbilical in small-gut obstruction, and hypogastric in obstruction of the large gut.

In acute cases (especially in strangulation) *shock* is evidenced by pallor, sweating, sub-normal temperature, cold extremities, shallow respiration and feeble pulse. Shock is not a feature of chronic obstruction unless an acute attack supervenes.

Vomiting is nearly always present in greater or less degree. It is earlier, more frequent and more copious the higher up in the intestinal canal is the obstruction. Stoppage in the upper jejunum causes copious bilious vomit, but obstruction of the large bowel is accompanied by only a slight degree of vomiting. Commonly, in obstruction of the small intestine the vomiting has a definite sequence. First the stomach contents are returned, then bilious vomit is brought up, and finally yellowish-brown *fæculent* material is ejected from the stomach in large quantities. *Fæculent* vomit in the absence of a gastrocolic fistula is pathognomonic of intestinal obstruction.

Constipation, though a necessary consequence of intestinal obstruction, is not always immediately evident. An enema may bring away *fæcal* material from the lower bowel, and occasionally there may be a natural emptying of the bowel after obstruction high up has occurred. Barnard's suggestion to administer two turpentine enemata, the first to empty the lower bowel, the second four hours later to determine whether obstruction be present, is a good one.

Distension ensues always sooner or later. In *volvulus* it usually comes on very quickly, in most other forms of obstruction more gradually. In large-gut obstruction the distension may develop to a considerable degree before the acute symptoms show themselves. In obstruction low down in the small gut the obstructed coils often show through the abdominal wall in the characteristic "ladder pattern."

Distension is often local at first. General abdominal distension is never to be waited for when there are unequivocal symptoms of obstruction.

Visible peristalsis is almost diagnostic when present, but it is only in a patient with a thin-

INTESTINAL OBSTRUCTION

walled abdomen that the peristaltic wave can be seen to move along the intestine.

Abdominal tenderness can sometimes be elicited when there is distension, and in some cases there may be *rigidity of the abdominal wall* over the site of distended tender coils. But rigidity is neither a constant nor a usual feature.

The late symptoms of obstruction are often those of peritonitis and toxæmic shock.

Types of obstruction.—Intestinal obstruction may be acute or chronic. Three main types of **acute obstruction** can be recognized, according to the level at which obstruction exists.

1. *Obstruction in the upper part of the small intestine.*—In these cases shock is great, vomiting frequent and copious and early in onset, and these acute symptoms may entirely overshadow and precede distension and constipation.

2. *Obstruction of the lower part of the small intestine.*—Here shock is still noticeable, vomiting ensues after a few hours and the vomit gradually changes till it becomes fæcalent, distension soon comes on and is sometimes of the "ladder pattern," constipation is present, and peristalsis may sometimes be seen.

3. *Large-bowel obstruction* is unattended by shock (save in some cases of acute volvulus and intussusception), does not give rise to much vomiting, is accompanied almost from the first by severe if not absolute constipation, and in many cases distension is very advanced before the general symptoms become acute.

In **chronic obstruction**, gradually increasing constipation is the main symptom. It is attended by attacks of colicky pain and possibly local distension. Attacks of diarrhoea may alternate with the bouts of constipation.

Diagnosis.—This must be considered in two parts—the diagnosis (1) of acute and (2) of chronic cases.

1. **Acute variety.**—Here the diagnosis is usually clear in cases where the small gut is involved. When a patient is seized with acute epigastric abdominal pain and becomes collapsed, with feeble pulse, cold extremities, anxious look, and sweating skin, and soon begins to vomit first the stomach contents, then bile, then darker material which becomes fæcalent, while the abdomen remains flaccid and tender, he is suffering from acute obstruction of the *small intestine*. The diagnosis should be made, if possible, before distension appears, and it is not always necessary to demonstrate absolute constipation.

In the *differential diagnosis* of acute small-gut

obstruction all the hernial orifices must first of all be examined. When no distension is present, acute obstruction has to be distinguished from all the other acute abdominal catastrophes. From the *acute inflammations*—perforated gastric ulcer, pancreatitis, appendicitis with peritonitis, cholecystitis—it is differentiated by the absence of rigidity, and by the more frequent vomiting, which becomes fæcalent; from *renal or biliary colic*, by the location and radiation of the pain. In colic the vomiting may be severe and collapse great but there is never fæcalent vomit, and distension does not follow, nor is constipation constant. In *torsion of a viscus*, vomiting comes on early but does not become fæcalent. *Gastric crisis* is excluded by finding no other sign of tabes.

In *acute obstruction of the large bowel* the pain is not so great, the vomiting not so frequent, and the shock much less. Distension, however, is usually a more evident feature.

In cases of small-gut obstruction in which distension has developed and in acute obstruction of the large bowel in which distension is an early sign, it is necessary to exclude mesenteric thrombosis or embolism, uræmia, and the late stages of peritonitis from any cause. *Uræmia* should be diagnosed by careful consideration of the history and by examination of the urine for albumin. It is probably impossible to distinguish between a late case of intestinal obstruction and cases of *mesenteric thrombosis or embolism*, or *late peritonitis*. But in late peritonitis the history should give some indication. In the latter condition there is usually paralytic or mechanical obstruction, whilst in late obstruction there is frequently some peritonitis; in peritonitis, however, the vomiting is seldom fæcalent, as is commonly the case with late obstruction.

Strangulation of an external hernia.—This is the most frequent cause of acute obstruction, is the most easily diagnosed variety, and yet is responsible for more deaths than any other single cause. The reason for this is probably that the very visibility of the hernia deceives the observer as to the seriousness of the obstruction, and attempts at taxis are continued too long. More lives would be saved if taxis were never attempted on a painful tense hernia, but every such case promptly handed over to the operating surgeon.

2. **Chronic obstruction of the small intestine** causes recurrent attacks of acute abdominal pain and vomiting. The attacks tend to become more frequent, and ensue soon after the

INTESTINAL SAND

taking of food. Gradual loss of weight occurs. Radiography after the administration of an opaque meal would serve to show the obstruction. Exploration of the abdomen is sometimes necessary for diagnosis.

Chronic obstruction of the *large bowel* is usually due to cancerous stricture or diverticulitis. The subject is considered under *INTESTINE, NEW GROWTHS OF*.

Treatment.—For the operations appropriate to the various forms of intestinal obstruction the reader must be referred to works on operative surgery.

ZACHARY COPE.

INTESTINAL SAND (*Sable intestinal*).—

The passing of gritty particles by the bowel. There are two varieties—the true or inorganic, which consists of the phosphates or carbonates of calcium and magnesium; and the false or organic, comprising indigestible particles of food, especially the sclerenchyma of pears and other fruits. Intestinal sand may be passed intermittently for several months in cases of mucous colitis.

FREDERICK LANGMEAD.

INTESTINAL TUBERCULOSIS (*see* *INTESTINE, TUBERCULOSIS OF*).

INTESTINAL WORMS.—The worms which infest the human intestine will be considered in the following order:—

ENTEROBIUS (OXYURIS) VERMICULARIS.
TRICHURIS TRICHIURA (*TRICHOCEPHALUS* DISPAR).

ASCARIS LUMBRICOIDES.
ANCYLOSTOMA DUODENALE.
NEMATODUS AMERICANUS.

TÆNIA SAGINATA.
TÆNIA SOLIUM.

DIPHYLLOBOTRIUM LATUM (*DIBOTRIODIPHYLLOCEPHALUS LATUS*).

ENTEROBIUS (OXYURIS) VERMICULARIS
(*PLATE 15, Figs. 1, 1'*)

This species, commonly known as the thread-worm or seatworm, is very small. The male measures 3-5 mm. in length by 0.1 mm. in breadth, the female 10 mm. by 0.5 mm. in breadth. The males are seldom seen. The sexes are readily differentiated, the male having a truncated and the female a pointed tail. The eggs are oval (0.05 mm. long by 0.02 mm. wide), with a thin shell consisting of three layers (*PLATE 15, A*). Primary infestation is due to their ingestion on fruit, vegetables, or other foods which have been touched by

INTESTINAL WORMS

infected hands, and direct autoinfestation from dirty fingers is extremely common.

The worms inhabit the whole of the large intestine, including the appendix, and the adult females sometimes leave the anus in the evening. This causes itching, to relieve which the patient scratches himself and so gets eggs under the finger-nails and spreads them over the surface of the body. By picking the nose or sucking and biting the fingers—habits common in infected children—the eggs are conveyed to the upper part of the alimentary tract and a fresh generation of oxyurides is started.

They are one of the commonest parasites of man all over the world, but occur chiefly in children and young adults. They are usually present in large, often in immense, numbers.

Symptomatology.—The worms themselves produce no toxin, and do not injure the health in this way. Very large numbers may cause vague abdominal pain, or pain localized to the region of the appendix; and some irregularity of the bowels, with excess of mucus in the stools, is not uncommon. The appetite may become capricious. Local symptoms are produced by the nocturnal wanderings of the females, which often cause intense irritation. Children sleep badly and wake unrefreshed, and so suffer deterioration of health. Scratching the anus produces eczema; and in little girls, in whom the worms often wander into the vagina, a vulvo-vaginitis may be set up.

The irritation of the perineum is a common cause of nocturnal enuresis; and more serious still is the fact that masturbation in both boys and girls frequently commences as a result of their presence, and may continue after their destruction. Their presence will often prevent the cure of a chorea, tic, epilepsy, or functional nervous disorder. Convulsions also may be caused in ricketty infants.

Diagnosis.—Any of the symptoms just described occurring in a child should lead to an inspection for the parasites in the evening. The stools may also be examined for ova and adult worms.

Treatment.—This must be directed chiefly to prevent reinfestation. The child should wear drawers in bed to prevent scratching. The anus should be rubbed nightly with a mercurial ointment. The finger-nails should be kept short and clean, and picking the nose and biting the fingers should be prevented as far as possible. To get rid of the worms them-

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selves the simplest way is to use an enema of salt and water, a tablespoonful to the half-pint, or infusion of quassia.

If this is repeated nightly while the worms are numerous, and then at longer intervals, the patient will be free in six weeks, unless reinfestation occurs. Worms continue to mature for that period from a single infestation, and since the enemata do not reach the whole of the large intestine a more rapid cure is impossible.

TRICHURIS TRICHIURA (TRICHOCEPHALUS DISPAR (PLATE 15, Figs. 2, 2')

The whipworm is a very common parasite. It has a long filiform anterior half with a thicker posterior half. The female is about 50 mm. long, the male somewhat shorter. Their chief habitat is the cæcum, where they imbed their filamentous extremity in the mucous membrane. They are usually present in small numbers and cause no symptoms. Infestation occurs by food and water.

Symptomatology, diagnosis, treatment.

—In large numbers the worms may cause anæmia—for they feed on blood—and reflex symptoms of varying severity. Occasionally perforation of the intestine occurs. They may be recognized by the characteristic ova, which have a transparent projection at each end (PLATE 15, b). No anthelmintic is reliable.

ASCARIS LUMBRICOIDES (PLATE 15, Figs. 3, 3')

This species, the roundworm, somewhat resembles an earthworm in size and shape, being some 15–25 cm. long in the male, and 20–40 cm. long by 5 mm. wide in the female. The colour is reddish yellow and the surface roughened. The egg has a chitinous shell with a thick outer coating of albuminous material, irregularly nodulated and stained brown or yellow. The contained ovum is unsegmented. (PLATE 15, c.)

The worm occurs all over the world and infests people of all ages, though it is commonest in children in European countries. The individual rarely harbours many, but sometimes very large numbers are present. They live in the upper part of the small intestine, but sometimes wander; they have been found in the glottis, the nares, the bile-ducts, the peritoneum, and in the contents of a so-called verminous abscess.

The egg can live for many months after leaving the body. It is probable that infection occurs through the ingestion of eggs in water or

uncooked food without the intervention of any intermediate host.

Symptomatology.—In a majority of cases no symptoms are produced. In others, especially children, abdominal pain, dull or colicky, nausea, vomiting, and irregular action of the bowels may be caused. An illness simulating typhoid fever has been caused occasionally. The appetite may be poor, or ravenous, or perverted. Anæmia and sometimes slight eosinophilia may be found. Nervous symptoms, often severe, are not very uncommon in the more heavily infested cases; these include reflex irritation of the nose, deafness, dumbness, amaurosis, paralyses and other neuroses, epileptiform attacks, and tics. That they are produced by *ascaris* is proved by their being cured at once by the expulsion of the worms.

Diagnosis.—If one worm has been passed, others may be present. Apart from this, their presence can be suspected from the symptoms, and confirmed by examining the stools for the ova, which in infested individuals are present in immense numbers and can be recognized with ease. In doubtful cases there is no harm in giving a dose of santonin.

Treatment.—Santonin is the best remedy. It can be given with calomel or scammony, 2 gr. of each for a child of over 2 years of age, up to 5 gr. for an adult. It is very effective combined with castor oil, administered at bedtime, and followed in the morning by a second dose of castor oil. An overdose of santonin may cause purging and vomiting, or even convulsions and death, so that in very young and weakly infants it should be used with caution. It may cause yellow vision, and makes the urine a deep yellow, which becomes pink on the addition of a caustic alkali.

ANKYLOSTOMIASIS or UNCINARIASIS (PLATE 15, Figs. 4, 4', 5, 5')

Hookworm disease, as this affection is also called, is caused by infection with one of the two hookworms, *Ancylostoma duodenale* (*Uncinaria duodenalis*) and *Necator americanus* (*Uncinaria americana*). In England it is sometimes known as miner's anæmia.

Ancylostoma duodenale.—In both sexes the body is cylindrical, and white in colour, unless filled with blood, which imparts to it a reddish tinge. The males are 8–10 mm. long the females 10–12 mm. The mouth in both sexes is formidably armed, there are four strong curved chitinous teeth near the margin placed ventrally, and two placed dorsally, whilst at

PLATE 15.—INTESTINAL WORMS, WITH OVA

- 1, *Enterobius* (*Oxyuris*) *vermicularis*, male. × 8.
- 1', " " " female. × 8.
- 2, *Trichuris trichiura*, male. × 5.
- 2', " " " female. × 5.
- 3, *Ascaris lumbricoides*, male. $\frac{1}{2}$ nat. size.
- 3', " " " female. $\frac{1}{2}$ nat. size.
- 4, *Ancylostoma duodenale*, male. × 10.
- 4', " " " female. × 10.
- 5, *Necator americanus*, male. × 10.
- 5', " " " female. × 10.
- 6, *Tænia saginata*, head. × 7.
- 6', " " proglottis (mature). × 2.
- 7, *Tænia solium*, head. × 7.
- 7', " " proglottis (mature). × 2.
- 8, *Diphyllobothrium latum*, head. × 8.
- 8', " " fairly mature proglottis. × 10.

- | | |
|---|--------------|
| A, Ovum of <i>Enterobius vermicularis</i> . | } All × 400. |
| B, " <i>Trichuris trichiura</i> . | |
| C, " <i>Ascaris lumbricoides</i> . | |
| D, " <i>Ancylostoma duodenale</i> . | |
| E, " <i>Necator americanus</i> . | |
| F, " <i>Tænia saginata</i> . | |
| G, " <i>solium</i> . | |
| H, " <i>Diphyllobothrium latum</i> . | |

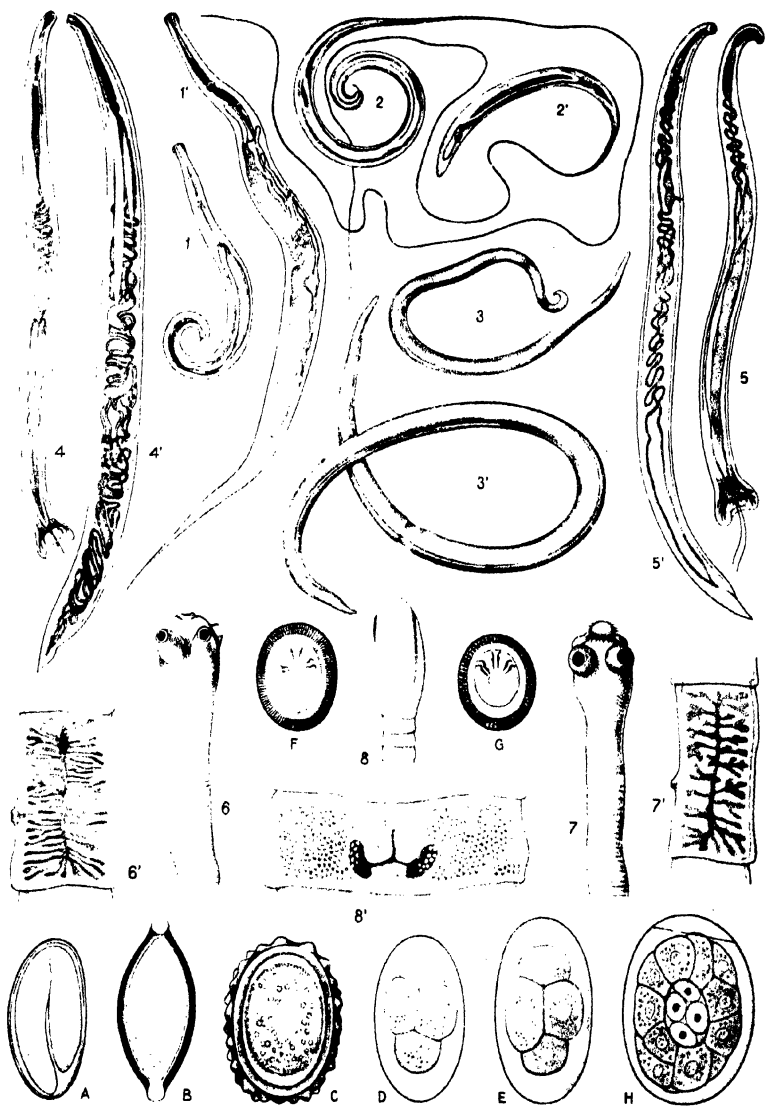


PLATE 15.—INTESTINAL WORMS, WITH OVA.

INTESTINAL WORMS

the base of the oral cavity lie a single conical projection on the dorsal side and two lamellæ of chitin on the ventral. The male worm becomes gradually broader towards the anal extremity and ends in a broad expansion, the genital bursa, which has two large lateral and a small dorsal process or expansion. In the bursa are two fine spicules.

The female terminates in a tapering extremity and the vagina opens at the junction of the middle and posterior thirds of the body, so that in copulation the sexes form the figure Y. The eggs are oval (0.06 mm. by 0.04 mm.), with a thin shell and segmented contents (PLATE 15, D). Enormous numbers are being continually laid by the mature females; they leave the body in the faeces. Under suitable conditions of warmth and moisture development proceeds with great rapidity, and in one to three days a rhabditiform larva, 0.21 mm. in length, emerges. The young larva is very active and feeds on organic matter, growing fast and changing skin twice. When about 0.6 mm. long it ceases to feed and becomes less active, but is much more resistant to changes in its environment.

Larvæ can live for several months in the moist earth, awaiting an opportunity to enter another host by piercing the unbroken skin of the hands or feet. It is probable that infestation never occurs by direct transference to the alimentary canal in food or water. From the lymphatics of the skin the larvæ proceed in the lymph-stream to the thoracic duct, and so into the venous system; or they may pierce a superficial venule, and reach it more directly. Passing through the right side of the heart they reach the pulmonary capillaries, and from these make their way into the air-vesicles. They then pass up the bronchioles, bronchi, and trachea, and, turning down into the œsophagus, pass through the stomach, and at last come to rest in the lower part of the duodenum or upper part of the jejunum. Before becoming adult they pass through three more stages, and at the end of the second of these acquire the hooked teeth. They are mature in a month. Great numbers often infest the same person, so that hundreds or even thousands are sometimes found in the small intestine. They often move their position, and the oozing of blood from the minute wounds may cause serious loss if many hookworms are present.

The geographical distribution of ankylostomiasis is very wide, and is only limited by the

susceptibility of the young larvæ to cold. The species inhabits the whole tropical and sub-tropical belt, but in temperate climes is only endemic in specially favourable localities. The best-known is the St. Gothard Tunnel, where the worm was first recognized as the cause of "tunnel disease." It is common in the Westphalian coal-mines and the tin-mines of Cornwall, where it produces the so-called miner's anæmia, and it has recently been imported into some of the British coal-mines.

Symptomatology.—Considerable irritation of the skin is set up by the entrance of numbers of larvæ, and a dermatitis known in Ceylon as "coolie itch" and in Cornwall as "bunches" is produced. The adult worms cause a train of symptoms due in part to loss of blood and in part to irritation of the alimentary canal. The earliest of the latter are epigastric tenderness and pain, relieved by taking food. The appetite is generally unnaturally large, but may be lost or perverted. Constipation is the rule, but sometimes there is alternate constipation and diarrhoea, apt to be accompanied by colicky pain. The loss of blood leads to the usual symptoms of anæmia—pallor, shortness of breath, faintness, palpitation of the heart, and general lassitude. The heart-muscle, insufficiently nourished, becomes fattily degenerated, and dilatation of the organ with loud hemic murmurs can be detected on physical examination. The patient remains fat. (Edema is common in the graver cases, at first confined to the feet, later becoming general, and accompanied by passive effusions into the serous cavities. In the dark races the pallor is best observed by making the patient put out his tongue, the extreme whiteness of which makes a striking contrast with the pigmented skin.

The worst cases sink rapidly and die from exhaustion. Less severe cases continue for a long time in a state of ill-health, and chronic infestation in children leads to infantilism with stunting of growth and delayed puberty.

The type of anæmia produced resembles that of chlorosis, and is of a high grade, with great increase in the volume of the blood. A count of less than 1,000,000 red cells per c.mm. is not unknown, whilst one of 1,500,000 is common. The hæmoglobin is reduced still more, and a low colour-index is produced. A film shows the presence of normoblasts in most cases, of megaloblasts occasionally, and of poikilocytes. There is often a leucocytosis, and in the more recent and acute infestations it may

INTESTINAL WORMS

reach up to or above 50,000 per c.mm. Eosinophilia is the rule, eosinophil cells averaging 10 per cent. and sometimes forming 50 per cent. of the total white cells.

Diagnosis.—Among coolies in the tropics there is a very high incidence of infection, often as high as 80 per cent., but only a small proportion of these suffer from the symptoms of ankylostomiasis, though all are a source of danger to others. Any anæmia, especially accompanied by digestive disorder, in one who lives in or has recently left the tropics should raise a suspicion of parasitism by the hook-worm. A blood-count should be undertaken, and anæmia of chlorotic type with eosinophilia would be strongly confirmatory. Definite proof can be obtained by examining the stools for ova. If they are present their numbers give an indication of the number of worms—150 per cg. of fæces represents about 1,000 worms. Eosinophilia and pronounced anæmia are more commonly due to ankylostomiasis than to any other condition.

Treatment.—The usual remedies are thymol, beta-naphthol, the two in combination, or oil of eucalyptus. The dosage depends largely on the age and condition of the patient. Thymol should be given in large doses, 15–30 gr., at 8, 10, and 12 noon, and a purgative should be administered if the bowels have not been opened freely before midnight. Great care should be taken to prevent absorption; alcohol in any form, glycerin, turpentine, and alkalis should be absolutely prohibited. Even with every precaution, cases of poisoning with delirium, collapse, and even death occur now and then. Beta-naphthol is safer and equally efficacious. To a strong adult 30 gr. can be given thrice at two-hourly intervals, followed in another two hours by 1 drachm of magnesium sulphate. In the worst cases and in children it is wiser to give 10 gr. once a day for a week instead. These methods should be modified for intermediate cases. The stools should be examined eight days after the treatment, and, if ova are still present, the course should be repeated.

With oil of eucalyptus the following mixture is a good one:—

Ry Oil of eucalyptus ℥xxx.
Chloroform ℥xl.
Castor oil 3x.

Give half the mixture on waking, and the rest half an hour later. Repeat on alternate days until the stools are free of ova.

Whatever treatment is adopted, the patient should be fed on easily-absorbed concentrated

food for two days before it is started, and an enema given. In the intervals between the separate courses of vermifuges, give iron for the anæmia and continue it afterwards until the blood is normal.

Necator americanus.—The American hook-worm closely resembles the common species in general characters, but differs greatly in the buccal armature. The oral cavity is small, with a ventral pair of semilunar cutting plates and a pair of less well developed dorsal plates of the same kind, and deep within are pairs of dorsal and ventral teeth of small size. The ova are larger and a little more pointed, but are smooth and have segmented contents. The life-history is incompletely known, but probably agrees with that of the *ancylostoma*. The adult worms live in numbers in the upper part of the small intestine. This is the prevalent species in North and South America, and also occurs in Africa and the Indo-Malayan region. The ova (PLATE 15, E) resemble those of *A. duodenale*. The symptoms, diagnosis, and treatment are the same as those of ankylostomiasis.

TAPEWORMS

Tania saginata (T. mediocanellata).—This tapeworm (PLATE 15, Figs. 6, 6') attains a length of 4–10 metres. The head is cubical, grey in colour, and measures 1.5–2 mm. in diameter. There are four powerful suckers. The neck is long and extensible and about 1 mm. wide. Behind the neck is the chain of proglottides, which become larger until those of full maturity are reached, the largest being some 16–20 mm. long by 4–7 mm. wide. The total number is about 1,000 to 1,500. A few are shed daily and escape singly in the fæces or make their own way independently through the anus. Each proglottis has a genital pore which opens laterally just behind the centre, and the openings show no regular alternation. Each leads to the uterus, which has 20–35 lateral branches, and these themselves branch again. The ova are very minute (0.03–0.04 mm. long by 0.02–0.03 mm. broad), slightly oval and brown, and have a thin transparent shell. Inside a thick striated embryonal shell lies the six-hooked embryo. (PLATE 15, F.) The adult tapeworm, usually solitary, lives only in the intestinal canal of man, sometimes for many years. The cysticercus or bladderworm stage is passed in the ox, and is known as *Cysticercus bovis*. After some months, if not ingested by man, it dies and becomes calcareous.

INTESTINAL WORMS

The cysticerci are very small transparent cysts (8 mm. by 5.5 mm.) with a minute central dot, the head. They are few in number and are found in the muscles, especially the pterygoids. Infestation in adults occurs through eating undercooked beef, in infants from feeding on raw-meat juice.

The tapeworm is of worldwide distribution, but in the temperate zone is most common in Eastern Europe and Asia, and, although rarer in England, is nevertheless our commonest tapeworm.

Tænia solium (PLATE 15, Figs. 7, 7') is smaller than *T. saginata*, being usually 2-3 metres long. The head is rounded, with four suckers, and a rostellum that bears 26 to 28 hooks, alternately small and large, in a double circle. The neck is long and thin. The proglottides number 800-900; the terminal ones are long and narrow, as in *T. saginata*, and measure 10-12 mm. by 5-6 mm. The laterally-situated genital pores usually alternate evenly and are placed just behind the middle of each segment. The uterus of the mature proglottis has a central trunk with 7-10 lateral branches, which themselves branch again. The eggs, minute and globular (0.03-0.035 mm. in diameter), have a thin shell; the embryonal shell is thick, radially striated, and yellowish in colour. Within is the six-hooked embryo. (PLATE 15, G.) The proglottides usually fall off in small chains and pass out in the fæces. The ova are expelled by the contractions of the proglottis, and are taken into the stomach of the intermediate host. The shell is dissolved in the gastric juice, and the free embryo pushes its way through the intestine into the viscera, muscles, or connective tissues, especially the intramuscular connective tissue. Here a small clear oval cysticercus (8-10 mm. long) develops, within which lies the invaginated head of the worm. The parasite in this stage is the *Cysticercus cellulosæ*. The favourite host is the pig, but man is sometimes attacked. Cysticerci generally die and undergo calcification after a few months, but may live for years. The adult worm infests man alone through the eating of mealy pork, and several are generally present in one patient.

Diphyllobothrium latum (*Dibothriocephalus latus*).—The broad tapeworm (PLATE 15, Figs. 8, 8') is 2-12 metres or even more in length. The flattened oval head has two deep, sharp-edged suckorial grooves on each side. There are no hooks. The neck is long and thin, and the proglottides are short and broad (10-12 mm.

by 4-5 mm.). Even the most elongated are no longer than they are broad. The two sexual openings, close together, are placed near the centre of the ventral surface.

In ripe segments the uterus is much folded and forms the "uterine rosette." The eggs are relatively large (0.07 by 0.045 mm.), with a brown shell and segmented contents. (PLATE 15, H.) They are deposited before the proglottides become detached. The embryo leaves the egg after lying in water for some weeks. At first it is ciliated, swims actively, and has three pairs of fine hooks; later it escapes from its ciliated coat and then creeps along the mud. The first host is a cyclops or diaptomus, in which it passes the procercoid stage. This is eaten by the second host, some fresh-water fish—pike, perch, trout, and others—and the embryo settles down in the muscles, intestinal wall, liver, spleen, or sexual gland. It then elongates, loses its hook, and develops sucking grooves. The head and tail are invaginated. This, the plerocercoid, is resistant to smoking, salting, and pickling. The adult worm develops in man, the dog, and the cat from eating fish containing living plerocercoids, and can survive for many years, several generally dwelling in each host. The distribution is local, the chief centres being the shores of the Baltic, the Swiss lakes, Japan, and Turkey, but from eating exported fish isolated cases occur all over the world.

Symptomatology and pathology.—Often no symptoms are produced by tapeworms; in other cases there are digestive disturbances. The appetite may be ravenous or capricious, with nausea, vague abdominal pains, or even colic. The bowels are generally irregular. Anæmia is sometimes present, due to absorption of toxins, which also cause a transient eosinophilia. The dibothriocephalus occasionally causes a condition resembling pernicious anæmia. Nucleated red cells, chiefly megaloblasts, are present, and there are poikilocytosis and polychromatosis. Nervous symptoms of some severity, such as epileptiform attacks and hysterical manifestations, have been caused by tapeworms, but much less often than by roundworms.

The *Cysticercus cellulosæ* is met with in small numbers or in hundreds or even thousands. It is commonest in the brain and eye, rarer in the heart, liver, lungs, and subcutaneous tissues. In the brain it grows in the pia or arachnoid mater, reaches a diameter of 10-20 cm., and has many branches—*Cysticercus*

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racemosus. The symptoms are those of cerebral tumour. Exact diagnosis is seldom made in the absence of subcutaneous cysticerci in addition. These form small cysts about the size of a pea. In the eye they destroy the organ, in the heart they produce irregularity, and in the lungs a condition which resembles asthma.

Diagnosis is usually made by the patient discovering ripe proglottides in the clothing or motions. By examination of a ripe proglottis the species of tapeworm can at once be determined.

Treatment.—Though it is quite easy to injure a tapeworm sufficiently to get rid of most of the proglottides, it is difficult to dislodge the head, and, unless this is done, the worm will grow again and begin to cast off segments in about two or three months. Great care, therefore, is necessary. Fluid and concentrated food, such as lean meat, milk puddings, and jelly, must be given for two or three days before the vermifuges, so that there is no bulky residue in the intestine. Syrup of figs or some similar laxative must be given each morning, and an enema of soap and water each evening. The patient must remain in bed the day the drug is given. *Filix mas* is probably the best, especially for *T. solium* and *D. latus*. The liquid extract of male fern is usually given in capsules owing to its nauseous flavour; but a not unpleasant jelly can be made by using the following formula:—

℞ Liquid extract of male fern ℥iss.
Gelatin gr. lvi.
Glycerin ℥i.
Elixir of saccharin ℥v.
Oil of cinnamon ℥iss.
Water ℥ivss.

Fifteen minims of the extract to be given every fifteen minutes until four to six doses have been administered.

If no aperient action has taken place after two hours, a tablespoonful of castor oil should be given, and repeated in half an hour if necessary.

Pelletierine tannate or sulphate may be given in doses of 5–8 gr. in the early morning, followed by a tablespoonful of castor oil half an hour later. Koussou in doses of 5–8 oz. made from the fresh material is effective, but owing to the danger of vomiting with consequent infestation with the *Cysticercus cellulosæ* it should not be given for *T. solium*. Oil of turpentine 1 oz. with castor oil 1 oz., in an emulsion with yolk of egg, is an efficacious

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but unpleasant remedy. Oil of eucalyptus as given in ankylostomiasis is good.

For young children a smaller dose of male fern may be given, but the safest drug is an electuary of bruised pumpkin seeds 1 oz. pounded with sugar and honey.

Among the less common tapeworms are two very small species of *Hymenolepis* and a larger *Dipylidium*. *Hymenolepis nana* occurs chiefly in children and is usually present in huge numbers. There is probably no intermediate host. *H. diminuta*, a tapeworm of rats and mice, occurs in children; its intermediary hosts are various insects. The frequency of these parasites in the British Isles and N. Europe is doubtful. *Dipylidium caninum*, a parasite of dogs and cats, is occasionally found in children who are infested by lice and fleas containing the cysticerci.

For *Tania echinococcus*, see HYDATID DISEASE.

E. A. COCKAYNE.

INTESTINE, NEW GROWTHS OF.—

New growths of the *small intestine* may be considered very briefly. Benign tumours of this part of the bowel are rare pathological curios. The only one which is common enough to be of any importance is the adenoma which forms a pedunculated growth projecting into the lumen of the gut; such a growth may initiate an intussusception. Malignant tumours are also rare. Carcinoma (columnar-celled) is met with in the lower part of the ileum, and gives rise to obstruction of the lower small-gut type (see **INTESTINAL OBSTRUCTION**). The pain is usually more violent than in obstruction of the colon by a similar growth. Very rarely a constricting type of cancer may affect the jejunum.

Sarcoma, either round- or spindle-celled, may occur in young people, but the symptoms are not very characteristic until a tumour appears.

NEW GROWTHS OF THE LARGE INTESTINE

There are two varieties of new growth of the large-gut which are fairly common, one simple, the other malignant.

Adenoma of Lieberkühn's glands is the common simple tumour. It may occur singly or in numbers. The common rectal polyp of children is of the same type. When multiple the tumours may be studded along the whole course of the colon. They may be sessile but are frequently polypoidal. The **symptoms** to which multiple papillomata give rise are—(1)

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attacks of colicky pain due to irregular colonic peristalsis, (2) diarrhoea or the passage of blood and mucus, (3) tenesmus when the lower part of the rectum is involved, (4) tenderness along the course of the colon, (5) progressive anaemia due to the repeated small hæmorrhages. The **diagnosis** is soon settled by digital rectal examination and sigmoidoscopy.

Treatment.—Many of the lower polypi can be removed if an anæsthetic be given, the proctoscope inserted, and forceps passed in to grasp and pull down those that are accessible. Frequently, however, the whole course of the colon may be affected, and then, apart from the radical operation of excision of the colon (which is by no means free from risk to life), it is only possible to palliate the condition by injection per rectum of astringents or of a dilute solution of resorcin. Appendicostomy or cæcostomy and irrigation from above might in some cases be advised.

Cancer of the colon accounts for about one in every two hundred cases admitted to the surgical wards of a general hospital. It is most common in the sigmoid (iliac and pelvic) colon; then the frequency of incidence diminishes in the following order, viz. cæcum, transverse colon, splenic flexure, hepatic flexure, ascending and descending colon. (For cancer of the rectum, see RECTUM, NEW GROWTHS OF.)

Pathology.—The microscopic anatomy in practically all cases is that of a columnar-celled carcinoma, which arises from the glands of Lieberkühn. Wide variations exist in the reaction of the connective tissue to the invading cancer-cells. If there is but slight resistance to their advance a tumour forms by the extension of the growth both along and round the bowel. This form is prone to degenerative and ulcerative changes. In other cases the cancer is strenuously resisted by the formation of connective tissue, which contracts and strangles the advancing cancerous cells. This progress is comparable to the gradual advance of an atrophic scirrhus of the breast, with the important difference that the breast complaint may not cause any great inconvenience for years, while the bowel lesion tends to obstruct the lumen exactly in proportion to the natural attempts at cure. The lymphatics and blood-vessels course round the bowel-wall in a circular fashion, so that a ring of involved tissue is soon formed. Two main clinical types can therefore be recognized—the first with tumour-formation growing fairly rapidly; the second with no tumour to be felt, but causing a hard,

malignant ring, which constricts the bowel. This ring growth is often very narrow, and sometimes it might seem as though a piece of string had been tightly tied round the bowel. By recalling the normal sacculations of the large bowel, one can easily appreciate that it may need a very careful search to discover the puckered ring, even when the hand is inside the abdomen. There is a third and rarer variety of cancer of the colon which deserves a passing mention—that is, a malignant growth that takes origin from a simple adenomatous polypus.

The progress of cancer of the colon is usually comparatively slow. It has been stated to be the least malignant of the carcinomata. For a long time the disease is limited to the bowel, and the lymphatic glands are involved later than is the rule in most other parts of the body. One observer found that in 40 per cent. of fatal cases the disease was limited to the gut. This would probably prove to be an under-estimation of the frequency of gland-involvement if a larger series of cases were taken.

The local changes which result vary according to the type of growth. The first type, which grows rather quickly and forms a palpable tumour, is not so apt to cause intestinal obstruction; it is more cellular and softer than the second type, and although it may project into the bowel lumen so as to impede the advance of the contents, yet ulceration commonly minimizes or removes the obstruction. It is also more likely to spread to neighbouring organs and cause infiltration of adjacent viscera, such as bladder, kidney, and pancreas; infection of the ulcerated surface occurs, and the symptoms of colitis result.

The second type, or *annular scirrhus*, produces a slow but sure narrowing of the lumen of the gut, leading to hypertrophy immediately above the growth. This compensatory hypertrophy continues *pari passu* with the increasing obstruction until urgent symptoms arise from an attack of acute intestinal obstruction. It is a curious fact that in such an event the cæcum is usually the most distended portion of the gut, even though the obstruction be so far on as the sigmoid flexure; this distension may proceed almost to bursting-point. At least a partial explanation of this may be afforded by the researches of Cannon and others, who have shown that antiperistalsis is one of the normal movements of the ascending and transverse portions of the colon. It is possible that an organic obstruction may produce storms of

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antiperistaltic waves ending at the cæcum, which consequently becomes greatly distended.

Symptomatology.—The signs and symptoms differ according to the pathological characteristics of the cancer. There is no constant, ordered sequence of symptoms, for any one of a large number may be the first noticed. Constipation, diarrhœa, vomiting, pain, a tumour, increasing distension of the abdomen—one or several of these may herald the subsequent course. A point to be laid stress upon is that the condition may be latent, and an attack of acute or subacute obstruction may be the first serious symptom to which attention is drawn. This is especially true of the scirrhus type. It is quite common to meet with cases which furnish a history of only a few weeks' constipation in which the growth as seen at operation certainly must have been growing for a much longer period. It is therefore all the more necessary to pay attention to slight symptoms of bowel trouble, so that, if possible, any morbid condition may be detected in the earliest stages.

The symptoms may be classified under six headings, but it must be remembered that only one or two of the group may be present in any particular case.

1. *Symptoms due to bowel obstruction.*—These are pain, constipation, distension, nausea, vomiting, and visible peristalsis. The pain is due to the contractions of the gut above the narrowed part, and, like all violent contractions of circular involuntary muscle, is cramp-like or colicky in character. Usually it has no direct time-relation to the taking of food, but is often increased by taking aperients. In one patient whom I saw the taking of sweet things increased the pain. Very frequently the site of the maximum pain indicates the position of the growth. Hurst has shown that intestinal pain is chiefly due to distension; if we consider a peristaltic wave passing along the bowel, the pressure inside the lumen would certainly be greatest just above the obstruction, and in the colon, unlike the small intestine, pain is sometimes localized with fair accuracy. Sometimes a patient will describe it as travelling across the abdomen and increasing in intensity up to the site of maximum pain. During the early stages it is commonly mistaken for indigestion, and if accompanied by nausea or sickness is attributed to a "bilious attack." Vomiting is an infrequent occurrence until rather late, for the large bowel is much more tolerant of obstruction than the small intestine.

Gradually increasing constipation is often the first abnormality, and if this supervenes in a person over middle age who has previously been perfectly regular as to the bowels, suspicion should be aroused and a thorough investigation carried out. By constipation is meant the abnormally infrequent or insufficient passing of formed fæces. The consistence must be ascertained, since the so-called spurious diarrhœa due to irritation may cause frequent but ineffective movements of the bowel. Alternating constipation and diarrhœa may thus occur. Occasional attacks of distension and flatulence are common. The distension may be limited to the region of the abdomen where the colicky pains are greatest. It may happen that the gradual increase in size of the abdomen may be the first symptom to cause alarm. Visible peristalsis may sometimes be watched through the abdominal wall, and local swelling may subside with a gurgling sound due to passage of flatus through the stricture.

All the above symptoms are frequently so slight as to be regarded as trivial, and completely neglected by the patient, so that it is a common event for an attack of acute intestinal obstruction first to compel attention.

2. *Symptoms due to bowel ulceration.*—Diarrhœa and the passage of blood and mucus may result from ulceration of the bowel. As a rule, the bleeding is an insignificant feature, but in rare cases quite profuse hæmorrhage may ensue, doubtless due to erosion of a vessel of some size. If the cancer is high up, the blood may be dark and resemble melæna. No doubt occult blood would frequently be found in the fæces if it were more commonly sought after. Mucus is frequently passed, and is probably due to irritation of the glands of the mucous membrane caused by the growth and by its attendant bacteria, which readily thrive either amidst the obstructed bowel contents or on the ulcerated surface of the cancer. The fact that mucous colitis is often merely a symptom of some more serious disorder deserves wider recognition.

It would appear that diarrhœa can result from two causes. The spurious diarrhœa, or the diarrhœa of constipation, has already been mentioned; in other cases, I believe, extensive ulceration at the site of the growth is sufficient to cause it even when no obstruction is present. Moynihan asserts that diarrhœa is more common with growths of the right colon. The fact that diarrhœa may occur should be remembered, for it may lead patients to assert

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that the bowels are regular. I know of one patient who suffered from diarrhoea for eighteen months before the disease was discovered.

3. *The presence of a tumour.*—A tumour is frequently felt. It may attain a considerable size before it is noticed. It is prone to break down into the bowel lumen, and consequently obstruction and pain are not so pronounced. In the early stages the tumour is often mobile, especially when situated in a part which has a long mesentery, as the transverse or pelvic colon; mobility also occurs with tumours of the hepatic flexure, which moves with the right kidney on respiration. Fixation occurs when any pericolitis supervenes.

4. *Symptoms due to extension to other viscera.*—These vary according to the organ or viscera involved. The cancer may implicate the kidney or bladder, and set up symptoms suggestive of stone or other disease liable to attack those viscera. It may infiltrate the stomach wall and cause simulation of gastric disease. Ulceration may cause fistulous connexion between a part of the colon and the small intestine or another part of the colon. It is uncommon to meet with serious extension to other organs.

5. *Symptoms due to pericolitis and peritoneal infection.*—Inflammation of the tissues round the colon may be consequent on ulceration and extension of a malignant growth. A local abscess may form and mask the primary condition. Sometimes perforation of the bowel suddenly takes place into the general peritoneal cavity, and diffuse and fatal peritonitis ensues.

6. *General symptoms* are absent at first. The patient often looks robust. Later, as the growth advances, loss of weight occurs, and anæmia and cachexia develop, but a large number of patients come to a fatal ending from obstruction before they have wasted to any noticeable degree.

Diagnosis.—Any patient who complains of the recent onset of symptoms which might be attributed to cancer of the colon should have a most complete examination (see ABDOMINAL TUMOURS, DIAGNOSIS OF). Bowel irregularity supervening after middle age should never be overlooked. After a careful history of the case has been obtained, the abdomen should be inspected to detect unusual fullness, distension, or visible peristalsis; palpated to find any tumour, and percussed in the flanks to see if there is any free fluid. It must be remembered that the transverse colon often sags down to the iliac fossa or pelvis, and that the sigmoid may

be on the right side of the middle line. Little help is gained by auscultation, since borborygmi are usually audible without the help of the stethoscope.

A digital examination per anum is imperative. A cancer of the pelvic colon may occasionally be thus felt, for it sometimes prolapses into the lower portion of bowel, which may be ballooned. This prolapse is apt to mislead as to the true distance of the growth from the anus. It is also important to make a bimanual recto-abdominal examination, for some small mobile growths of the pelvic colon can thus be detected.

Since a certain number of colon-cancers occur in that part which is suitable for endoscopy, some may be revealed at an early stage by the use of the sigmoidoscope. Endoscopy of the sigmoid is free from risk if due precautions be taken, and furnishes most valuable information. For the technique, see SIGMOIDOSCOPY.

Another valuable method of diagnosis is the taking of an X-ray photograph of the abdomen after the administration of a bismuth meal. Any stricture is beautifully demonstrated, for the bismuth mush becomes massed on the proximal site of the obstruction, and a thin line may indicate its slow progress through the stricture. It has been found experimentally that food takes about four hours to reach the cæcum, and from this it takes about two hours to traverse each of the ascending, transverse, and descending parts of the colon. If it is thought that the obstruction is in the lower sigmoid, then the bismuth emulsion can be administered as an enema given in the knee-elbow position, and possibly through the sigmoidoscope. This method of diagnosis may enable one to diagnose the obstructing form of colonic cancer at an early stage. (See also X-RAYS, DIAGNOSTIC USES OF.)

In spite of all these methods, there will remain some cases which present indefinite symptoms and in which diagnosis is doubtful. The question of an exploratory abdominal section then arises. If the general condition is good enough, this is a justifiable and necessary procedure, and should be strongly urged upon the patient; but in constitutionally feeble subjects it is not wise to open the abdomen unless the indications are very definite.

Differential diagnosis.—The conditions from which cancer of the colon has to be distinguished are numerous. The early symptoms of obstruction, colicky pain, flatulence, and occasional vomiting, are frequently ascribed to

dyspepsia. When a more acute attack of obstruction supervenes, several conditions need to be excluded. Some help is afforded by the fact that, excluding strangulated hernias, cancer of the colon is the commonest cause of acute obstruction in persons over middle age. **Volvulus** is likely to give rise to more acute symptoms, and the distension to come on more rapidly. Subacute volvulus can rarely be diagnosed from cancer of the colon before operation. **Intussusception** in the adult is rare, and when it does occur is sometimes caused by a malignant growth. **Simple stricture** of the colon cannot be diagnosed from cancer before operation; indeed, a microscopical investigation is sometimes needed to establish the distinction. It is well known that symptoms due to obstruction of the large intestine are much less acute than those resulting from a similar obstruction of the small gut. Vomiting is less pronounced and comes on later, while pain is usually much less acute. This is what one would expect, for the fluid contents of the jejunum and ileum pass along quickly at the rate of 5 ft. an hour, while the colon usually retains its more solid contents for many hours, and may do so in some cases even for days or weeks without any serious consequences.

There is sometimes an intermittence in the acute obstructive symptoms caused by cancer. Those cases that come under observation with a tumour may be rather puzzling. In the cæcal region such a lump may be mistaken for *chronic appendicitis* or *hypertrophic tuberculosis of the cæcum*. A careful history should distinguish the former, but the latter may be indistinguishable from cancer unless tubercle bacilli are discovered in the faeces, or one of the tuberculin tests gives a positive indication. Absolute diagnosis is not so important, since excision is probably the best treatment for hypertrophic tuberculosis. At the hepatic flexure tumours of the kidney, liver, and gall-bladder have to be excluded. In the transverse colon and splenic flexure the tumour may be mistaken for gastric carcinoma, but *anæmia* and loss of weight are greater in the latter, and the pain has direct relation to the taking of food. Analysis of a test meal is important.

When the cancer invades or becomes adherent to a neighbouring viscus it causes symptoms of a misleading nature. All or some of the many methods of diagnosis in vesical or renal diseases can be utilized if symptoms are referred to

those organs; if the stomach is invaded it may be very difficult, apart from the history, to say in which part the disease began.

When general peritonitis ensues on the perforation of the gut, diagnosis of the cause may not be made till the abdomen is opened; when localized pericolicitis causes abscess-formation on the right side, it may easily be mistaken for an appendix-abscess.

Prognosis.—The prognosis of cancer of the colon is bad unless the condition is treated by operation. A fatal result follows from acute obstruction, involvement of other viscera, general peritonitis, or progressive *anæmia* and *asthenia*. The less malignant the growth, or the more the cancer is resisted locally, the sooner the obstructive symptoms arise, but the better the outlook for operative measures.

Treatment.—The treatment of cancer of the colon is surgical save for those cases which have gone too far for excision and present no obstructive symptoms. We might possibly also except some low sigmoid growths to which radium can be applied by means of the sigmoidoscope. The operation varies with the stage of the disease and the state of the patient when brought under observation. Speaking generally, the surgeon either excises or short-circuits the growth, or performs colostomy.

ZACHARY COPE.

INTESTINE, RUPTURE OF (see ABDOMINAL INJURIES).

INTESTINE, TUBERCULOSIS OF.—Tuberculosis of the intestine, apart from tuberculous peritonitis (see PERITONITIS, TUBERCULOUS), for the most part comes into prominence owing to the symptoms of obstruction which it causes.

Narrowing of the gut-lumen may be caused by contraction of the scar-tissue resulting from the healing of a tuberculous ulcer, or by the formation of new tissue in the hyperplastic form of tuberculosis. The former is met with in the small intestine, the latter chiefly at the ileo-cæcal junction. It will be remembered also that in tuberculous peritonitis and *tabes mesenterica* obstruction of the bowel (usually the small bowel) may be due to adhesions, to pressure of caseous glands, or to the kinking of the gut.

When obstruction of the gut occurs as a result of tuberculous enteritis in the small gut, the history is usually sufficient to indicate the

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cause of the trouble. The obstruction will conform to the type of upper or lower small-gut obstruction according to the site of the constriction. *Ileo-cæcal hyperplastic tuberculosis* is a characteristic condition. As the name suggests, tuberculosis, when it attacks the cæcum and end of the ileum, causes a great tissue reaction. Fibrous and sometimes partially calcified tissues are thus formed, which project into or constrict the gut-lumen and cause an increasing obstruction. Adenomatous polypi may form in the interior of the gut over the site of the disease. Naked-eye inspection of the affected gut would on some occasions scarcely serve to distinguish the lesion from cancer or diverticulitis. Microscopically, the affected tissue will be found to contain giant cells with a large tissue-reaction. Tubercle bacilli may be detectable.

The **signs and symptoms** of ileo-cæcal tuberculosis may be described under four heads: (1) The passage of blood and mucus due to ulceration of the bowel may be noted. Occasional attacks of diarrhoea may occur. (2) A swelling in the right iliac fossa can be felt as the tuberculous mass grows larger. (3) The general health suffers, and wasting occurs from malnutrition. (4) Obstructive symptoms sooner or later supervene. At first these consist of occasional attacks of pain and constipation which may be taken for simple dyspepsia. Sooner or later, however, a more acute attack of severe colicky pain, vomiting, and distension is likely to call attention to the seriousness of the condition, and then the lump in the right iliac fossa will be noticed, even if it had escaped observation before.

Diagnosis.—Ileo-cæcal tuberculosis gives rise to symptoms almost indistinguishable from carcinoma or diverticulitis of the same region. If tubercle bacilli were discovered in the fæces, that would point to the nature of the disease, but it must be confessed that the condition is sometimes operated on, and the mass excised and microscopied, before the diagnosis can be made with absolute certainty.

Treatment.—It is unwise to treat the condition by medical means when a swelling can be made out in the right iliac fossa, or if there are any definite symptoms of obstruction, for before operation it is impossible to make sure that the condition is not cancer. Surgical measures are, therefore, to be advised. Abdominal exploration and excision or short-circuiting of the diseased portion of the gut is to be recommended.

ZACHARY COPE.

INTUSSUSCEPTION

INTRACRANIAL HÆMORRHAGE (see HEAD INJURIES).

INTUSSUSCEPTION.—*Acute* intussusception is the commonest cause of intestinal obstruction in infants. The majority of cases are met with in babies under 2 years of age, though the condition may be encountered at any age.

Etiology.—In adults an intussusception is commonly associated with a polypoidal growth of the intestine which excites vigorous gut-peristalsis; as the gut tries to force on the growth it drags and invaginates a portion of the intestinal wall. It is probable that in infants an indigestible bolus of food may provoke a too vigorous peristalsis in like manner, for it is a noteworthy fact that the maximum incidence falls between the sixth and ninth month of life, when weaning is usually in progress and portions of solid food may injudiciously be given to the infant. Intussusception usually occurs in infants who up to the time of illness have been very strong, well nourished, and healthy.

Pathology.—An intussusception consists in an invagination of one portion of bowel into the portion next to it. It is most common in the ileo-cæcal region, where the narrow ileum can more readily slip into the lax cæcum. The invaginated part continues to advance owing to the peristaltic movements of the gut. The part which is first invaginated remains at the apex of the intussuscepted portion, which may ultimately protrude at the anus. In an intussusception there are three layers, the outer ensheathing layer (the intussusciens), and the entering and returning layers of the intussuscepted portion (the intussusceptum). The vessels of the mesentery taken in with the intussusception become constricted and cause congestion and œdema of, and interstitial hæmorrhage into, the invaginated part. Ultimately the intussusception may become completely gangrenous. Natural cure has in rare cases been known to occur as the result of the casting off and passage per anum of the gangrenous part.

Varieties.—When an intussusception involves only the small intestine, it is termed *enteric*; when only the large intestine, it is known as *colic*; while *entero-colic* denotes that both ileum and colon are involved. The entero-colic variety is subdivided into the *ileo-cæcal* type, in which the apex of the invaginated part is the ileo-cæcal valve, and the *ileo-colic*.

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type, in which the end of the ileum forms that apex. Enteric and colic varieties are both uncommon.

Symptomatology.—Pain, shock, vomiting, the passage of blood and mucus per anum, and the presence of a tumour are the main clinical features. Distension is a late symptom which is of bad augury, and is never to be expected in the early stage at which diagnosis should be made. There is usually constipation. The main symptoms are pain, the passage of blood and mucus, and the presence of a tumour.

The pain comes on suddenly in a child who has previously been very well. It is accompanied by crying or screaming, by a drawing up of the legs, and by symptoms of shock. The bouts of pain last for a few minutes, and then there is a respite until the next attack. The shock is demonstrated by the sudden pallor of the face, by dilated pupils, and by an expression which can be readily interpreted as that of pain and apprehension.

Blood and slime begin to pass per anum within a few hours of the onset. The blood is not usually great in amount; sometimes it is mingled with a little faecal material. By the time blood is being passed per anum there is usually a swelling to be made out in the abdomen. It is sausage-shaped, is situated either in the right hypochondrium, epigastrium, or left lumbar region, and can be felt to harden as the attacks of pain (corresponding to peristaltic contraction) occur.

The abdominal wall is often rigid during the attacks of pain, so the swelling must be sought for between the bouts, when the muscles are flaccid and palpation is more easy. The absence of the caecum from the iliac fossa can usually be determined in entero-colic intussusception.

Vomiting is not usually an early symptom, nor is it ever a very notable feature unless and until distension and peritonitis are present.

Constipation is the rule, to which, however, there are important exceptions. The blood and mucus are sometimes mingled with faecal material, since in an early intussusception the occlusion of the bowel lumen is not always complete. A subacute and small intussusception may coexist with the passage of almost normal stools. The late symptoms of intussusception are those of complete intestinal obstruction and peritonitis. Distension increases, vomiting becomes more frequent, and toxæmia evident. The apex of the invagin-

ated part may protrude at the anus, and in some cases where that does not occur the advancing part may be felt on digital examination of the rectum. When the apex is not palpable per rectum, a ballooning and soft œdema of the bowel are sometimes detectable, indicating that the intussusception is just above the part palpated.

Diagnosis.—Very acute screaming attacks in a baby previously healthy, followed in a few hours by the passage of blood and mucus per rectum, almost always indicate intussusception. If, in addition, an oval tumour can be felt in the anatomical line of the colon, the diagnosis is assured. If the condition is suspected, but no tumour can be felt, it is necessary to give the child an anæsthetic and make a thorough examination with one hand on the abdomen and a finger of the other inserted into the rectum. By this means any abdominal swelling will be detected. An infant suspected of having an intussusception should never be left overnight with an undetermined diagnosis.

In entero-colic cases great help may be given by determining the absence of the caecum from the right iliac fossa.

In infants the **differential diagnosis** has to be made chiefly from simple colic and colitis. *Simple colic* is usually related to constipation or to the taking of some food which might cause intestinal disturbance, is transitory, and is relieved after the bowels have been opened. It is seldom accompanied by the passage of blood per anum, and no tumour can be felt in the abdomen. *Colitis* gives rise to most difficulties in diagnosis; but it is often preceded or accompanied by diarrhœa, and not by constipation or any symptom of obstruction. The colon may also be tender all along the affected part, and on palpating the right iliac fossa the caecum can usually be readily identified. There should be no abdominal tumour in colitis.

When an intussusception has protruded at the anus, diagnosis has to be made from a *prolapsed anus*. This is usually easy when the history of onset is carefully ascertained and the local swelling examined. In a prolapse of the anus the finger or a probe cannot be inserted between the external swelling and the external sphincter, whilst in prolapsed intussusception it can be so inserted in front of and laterally to the protruding mass. The opening of an anal or rectal prolapse is central to the projection, whilst in prolapsed intus-

susception it is at the posterior portion of the projecting apex.

That rare condition *Henoch's purpura* sometimes simulates intussusception, but the former generally occurs in older children and is usually accompanied either by joint manifestations or by purpura, which give the clue to the intestinal hæmorrhage and abdominal symptoms.

Subacute or chronic intussusception.—There are cases of intussusception in infants, and relatively more in older persons, which are subacute in onset and chronic in duration. In some instances symptoms may persist for several weeks before the acute symptoms of obstruction supervene. There are usually bouts of colicky abdominal pain, some irregularity of the bowels, and the presence of an abdominal swelling along some part of the line of the colon. Such cases may be mistaken for tuberculous peritonitis. In any case of doubt it is advisable to recommend the exploration of the abdomen, which would in any case be likely to do good.

Treatment of intussusception.—If left to itself the condition is nearly always fatal. Cases have been recorded in which reduction has been said to follow the injection of water or air into the rectum under low pressure. Such a method is unreliable and dangerous in that it may delay the adoption of the best method of treatment, which is manual reduction of the invagination after abdominal section

ZACHARY COPE

INVOLUTION OF UTERUS (see PUERPERIUM, COURSE AND MANAGEMENT OF).

IODINE POISONING (see POISONS AND POISONING).

IODISM (see DRUG ERUPTIONS).

IONIZATION (*syn.* Cataphoresis). Much work in this branch of electro-therapeutics may easily be carried out by the general practitioner with comparatively simple apparatus. A battery of about 24 dry cells is required. This should be fitted with a cell collector or arrangement for increasing the current by throwing into circuit individual cells without interrupting the current. A dead-beat milliamperemeter should also be in circuit. Various electrodes are also necessary, and rheophores or flexible wires to connect the electrodes with the battery. Solutions of the drug to be used

are generally of the strength of 1 or 2 per cent., and must be freshly prepared with distilled water. With such an outfit one may carry out the satisfactory treatment by ionic medication of conditions such as small rodent ulcers, warts, naevi, ozena, rhinitis, chronic gouty joints, "chronic rheumatism," and thickened or adherent pleura.

Care must be exercised in selecting the particular ion to be used, and also the right pole of the battery to drive the selected ion in. I have known a case to be treated for quite a long period by ionization with potassium iodide solution when it was desired to use the ions of iodine. The part treated was wrapped in lint soaked in a solution of that salt, and the positive pole of the galvanic battery was always attached to this, the negative pole being in a water-bath in which the patient placed his feet. Now, what happens in such circumstances when the current is turned on to a sufficient degree? The KI in the solution in the lint is electrolysed and decomposed, and the ions of potassium liberated, being kations, i.e. ions liberated at the positive pole (anode), and being charged with positive electricity and, therefore, repelled by the positive pole, seek the negative pole (kathode). The only way to the negative pole in this case was via the tissues which they entered. The liberated iodine ions which it was desired to use, being anions, that is, particles charged with negative electricity and seeking the positive pole, reached it on the outer side of the soaked lint without passing to the tissues. The current in this case should, of course, have been used in the reverse direction.

Ionic medication offers a means of affecting a particular part of the body by the drug used, the ions penetrating and permeating the tissues and entering the substance of every cell through which the current passes. In this way, a small quantity has a much greater effect than when applied in any other way.

The drug is eliminated at a much slower rate than when administered by the mouth or by subcutaneous injection; its effect is therefore much more lasting. We ought not to assume that the effects of drugs introduced into the system electrically, in the ionic form, are identical with those produced by administration through the mouth or by subcutaneous injection; for when administered in the ordinary way the drugs are added to the constituents of the body, whereas when administered electrically they are substituted for the normal

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ions of the tissues. It is, therefore, a method of substitution rather than of addition.

The effects of the transmission of ions into the tissues are of two kinds, viz., general and local.

With regard to general effects, Prof. Leduc has shown that the ions of strychnine and cyanogen can be introduced into the body of a living animal in poisonous doses, and has been able to produce symptoms of poisoning in his own person by means of morphia electrically introduced.

When it is necessary to administer a considerable dose of a drug, as in the case of iodine, this may be done by employing limb baths as electrodes, with which currents of 100 ma. or more can be used.

Experimental proof has been given by Frankhausen that ions can be made to leave the body in this way, and thus injurious ions may be extracted from the body. Poisonous metals have been so eliminated. Bordier has shown that the ion of uric acid can be extracted in considerable quantities by means of an arm bath used as the anode of an electric circuit, and it has been suggested that the same may be true of toxins.

Care should be taken to employ pure solutions when the effect of any special ion is desired, as otherwise the effect looked for may not be obtained. The solution should be made up with distilled water, and the material used as coverings of the electrodes should contain no foreign substances. Absorbent cotton-wool or pure lint will answer the purpose well. When it is desired to use the metallic ions of a salt, the plate forming the positive pole should, if possible, be the same metal as that of the salt. If this is not possible, pure carbon may be used, if the soaked pad is sufficiently thick to prevent the case from being complicated by the introduction of ions liberated from the metallic surface. It is sometimes convenient to place next the skin, under the electrode, a layer of gutta-percha tissue, having an opening cut in it corresponding to the size and shape of the area which it is desired to treat, and thus to circumscribe the area and strictly localize the effect to that area.

Convenient tablets of certain drugs can be procured which, when dissolved in 1 oz. of distilled water to each tablet, give a 1-per-cent. solution. Only such chemical substances as undergo dissociation or ionization when dissolved in water can be used.

Some organic medicinal substances are unsuitable for the purpose, because they are either not soluble in water, or do not dissociate when so dissolved, e.g. creosote. In addition to inorganic salts, there are many organic compounds which are soluble and dissociate and may be used with advantage in this way, such as the alkaloids, salicylic acid, aniline hydrochlorate and other coal-tar compounds which ionize in watery solution.

Chloroform, ether, chloral, and carbolic acid cannot be used. Neither should tinctures of barks, leaves, or roots, etc., of the Pharmacopœia be employed.

Superficial lesions lend themselves most readily to this form of treatment, as penetration is not rapid or deep. Radium ions, however, have been found as deep as 9 cm. from the surface after ionization with solution of radium bromide, even when the circulation of the part had been temporarily arrested.

If dilute simple acid solutions are used, the effect on the skin at the anode is that which is due to the introduction of the hydroxyl ions. This is the same for all ordinary acids.

In the case of dilute alkaline solutions, we get the effect of the introduction of the hydroxyl ions at the kathode. Protracted and strong applications produce a sore in each case which has its own peculiar characteristics. The ions of Na, K, or Mg do not produce very definite effects except when introduced in considerable amount; whilst the ions of the metals of the alkaline earths, on the other hand, if used in sufficiently strong doses, cause a surface destruction of the tissues which is characteristic.

Leduc reported that dogs experimented upon with solutions of calcium chloride were able to endure a current density of 15 ma. per square cm. for 30 minutes without any obvious discomfort. The surface at the anode appeared white, as though impregnated with calcium carbonate, sulphate, or phosphate. Inflammation and œdematous swelling soon set in, and the surface changed into an ulcer with undermined edges and an indurated base, which gradually healed, remaining indurated, however, for more than a month. Barium and strontium compounds were found to act in the same way. Most pain seems to be caused by the introduction of the carbonic acid radicle ion, one which exists in large amounts in the tissues of the body.

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A smooth, hard dryness of the surface seems to be the peculiar effect of the introduction of the sulphuric acid radicle ion.

The ions of many of the heavier metals are more or less caustic in their effects, coagulating albuminous substances. The zinc ion is an antiseptic of the first rank, and, when applied electrically, can be made to penetrate the tissues to any desired depth. Any wound or ulcer whose surface can be reached by the electrode may be disinfected by its employment. It does not produce any great inflammatory reaction. An ulcer caused by it in the skin of an animal shows no marked inflammatory effect nor sign of subsequent infection from germs, even if left completely uncovered, but remains aseptic, the contained ions of zinc appearing to act as the best possible antiseptic agent. Sinuses, fistulae, and chronic ulcers, including rodent ulcers, may be treated satisfactorily with them, the method being far preferable to curettage with its manifest risks of setting free infective agents and admitting them to the blood- and lymph-channels of the part curetted.

Copper ions have been used in the treatment of ringworm, but here the success has not been complete, there remaining scattered small islands of follicles that have not been sufficiently affected, and from these reinfection has taken place. Better results have been obtained by using 1 or 2 per cent. solutions of copper sulphate in treating rodent ulcer and lupus erythematosus. Sycosis has also been satisfactorily treated with copper ions, a copper wire being introduced into the suppurating follicles.

Magnesium ions may be used for multiple warts, a solution of the sulphate being in the pad of lint, to which is apposed a coiled flat magnesium wire spiral or a carbon electrode as the positive pole.

Chlorine ions are useful for dispersing fibrous tissue, producing resolution of scar formations. Fibrous adhesions about stiff joints can often be cured quickly without having recourse to the excessive passive movement which is so painful. This method, however, should only be used when the active inflammatory process which caused the stiffness has subsided. A 1-per-cent. solution of common salt is used as the electrolyte in a pad over the affected region, and the kathode or negative pole applied. A current of 20-60 ma. or more may be used. The knee, ankle, wrist, or elbow may readily

be treated in this way, as also may pleuritic adhesions.

Technique.—A case of a small rodent ulcer on the face may be taken as an example. The mode of procedure which I adopt is as follows: The area to be treated is first cleaned up, grease is removed, and a piece of oil silk, out of which is cut a hole the shape and size of the ulcer, is placed in position. A swab of absorbent cotton-wool soaked in a 2-per-cent. solution of zinc sulphate in distilled water is next applied to soak the tissues while the apparatus is being prepared for the actual treatment. The swab is then removed, and replaced by a pad of pure lint, about six layers thick, soaked in the 2-per-cent. solution. This is adjusted with as little handling as possible, and to it is applied one end of a zinc rod, such as is used for a wet Leclanché cell, this end of the rod having previously been scraped bright, under water. The wire at the other end of the rod is attached to the rheophore and connected with the positive pole of the battery. The negative pole is either applied to the patient in the form of a large wet flat pad between the shoulders, or is put, with the patient's arm, into a bath.

If the patient is wearing a dental plate, I make sure that this is first removed before commencing the actual treatment. The zinc rod is held steadily in position with a slight and uniform pressure while the current is gradually turned on and advanced till about half the milliamperage that we decide to use is indicated by the milliamperemeter. In estimating this, I allow 2-3 ma. for each square cm. in the hole cut in the oil silk. This half-strength current having been allowed to flow for five minutes, the current is gradually and without jerks advanced to the full strength determined on. The patient is then better able to stand this strength of current than he would have been if it had been applied during the first five minutes.

The current is allowed to flow continuously for half an hour and is then very gradually put back to zero. The zinc rod is held steadily in position during the whole of this time, and must on no account be removed before, or the break of contact will give the patient an electric shock.

On removal of the pad and oil silk, the area treated shows a pearly white surface, due to zinc ions having entered the tissues in their effort to reach the negative pole. Subse-

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quently there is some swelling and reaction lasting a few days and necessitating a simple soothing ointment dressing. A scab forms, and in about fourteen days this is thrown off and the ulcer has healed if it has been ionized sufficiently. If any ulceration remains it should not be treated until some two or three weeks have elapsed since the previous application.

E. S. WORRALL.

IRIDOCYCLITIS (see UVEAL TRACT, AFFECTIONS OF).

IRIDOPLEGIA (see OPHTHALMOPLEGIA).

IRIS, EXAMINATION OF (see EYE, EXAMINATION OF).

IRITIS (see UVEAL TRACT, AFFECTIONS OF).

IRRITANT POISONS (see POISONS AND POISONING).

ISCHIO-RECTAL ABSCESS (see ABSCESS, ISCHIO-RECTAL).

ITCH (see SCABIES).

JACKSONIAN EPILEPSY (see EPILEPSY, JACKSONIAN).

JACKSON'S MEMBRANE (see APPENDICITIS).

JAUNDICE.—Jaundice is due to the presence of bile pigment in the blood-serum, and is recognizable clinically by the yellow colour produced by its deposit in the conjunctivæ, skin, and mucous membranes.

It may be hæmatogenous, hæmo-hepatogenous (toxæmic), or hepatogenous (obstructive).

The existence of *hæmatogenous* jaundice is denied by some, but recent work has shown that although the liver is the chief seat of the transformation of hæmoglobin to bile pigment, the endothelium of the blood-vessels, pleuræ, and peritoneum can also perform this function. Hæmolytic jaundice is the best example of pure hæmatogenous jaundice. In this disease it is proved that there is no viscosity of the bile and no angio-cholangitis. Probably the jaundice in splenic anæmia, pernicious anæmia, and paroxysmal hæmoglobinuria also is partly hæmatogenous. Apart from these diseases, all jaundice is essentially obstructive.

Toxæmic jaundice is due to obstruction of the small intrahepatic ducts caused by cholangitis aided by increased viscosity of the bile. *Obstructive* jaundice is produced by mechanical blocking of the large bile-ducts. In these two varieties bile pigment is formed in the liver and escapes into the circulation by the liver capillaries and not by the lymphatics.

Diagnosis of jaundice.—The patient should be examined by daylight, as light

jaundice is difficult to detect by artificial light. The conjunctiva is the first and last part of the body to show an icteric tinge.

The following tests are useful in diagnosing and differentiating the different forms of jaundice:—

Urine. *Bile pigment.* (1) *Gmelin's test.*—Strong nitric acid in contact with urine shows a bluish-green ring and play of colours. (2) Tincture of iodine poured on to urine gives a green ring. When only a trace of bile pigment is present these tests fail, and it is necessary to precipitate the pigment with urates by adding saturated ammonium sulphate solution to the urine, filtering, and testing the residue with nitric acid.

Bile-salts. *Oliver's test.*—Dissolve peptone 30, salicylic acid 4, acetic acid 30, in distilled water 3,500 parts, and filter. Add urine 1 part to 3 parts of solution. Opalescence or a precipitate dissolving in acetic acid but not disappearing on boiling indicates the presence of bile-salts.

Urobilin.—This can be detected spectroscopically or by adding an alcoholic solution of zinc acetate (1 in 10) to urine, shaking, and adding a few drops of Lugol's solution. Urobilin causes fluorescence. (See also URINE, EXAMINATION OF.)

Fæces. *Stercobilin.*—Extract with alcohol; evaporate to dryness. Extract with water to which a little sulphuric acid has been added. Saturate with ammonium sulphate and dissolve the pigment in chloroform. With zinc acetate and ammonia a beautiful green fluorescence is given by stercobilin.

Liver insufficiency.—Ehrlich's tests for uro-

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bilinogen are described in URINE, EXAMINATION OF, and the galactose test under LIVER, CIRRHOSIS OF. The former is stated to be positive in tumours and amyloid disease, but the latter negative.

TOXÆMIC JAUNDICE

Etiology.—Toxæmic or hæmo-hepatogenous jaundice is due to the presence of some toxic body of chemical or microbic origin in the circulating blood.

In varying degrees the following results are produced: (1) Catarrh of the small intra-hepatic bile-ducts; (2) degeneration of the liver-cells; (3) increased destruction of red blood-corpuscles; (4) increased viscosity of the bile.

Examples of chemical poisons are tetrachlorethane, metadinitrobenzol, trinitrotoluene, toluylene-diamine, arsenobenzol compounds, arseniuretted hydrogen, chloroform, phosphorus, snake-bite and mushroom poisons.

The best-known microbic poisons are those of relapsing fever, syphilis, and spirochætosis icterohæmorrhagica, due to spirochætes; malaria and yellow fever, due to protozoa; pneumonia, pyæmia, septicæmia, and enteric fever, due to bacteria; influenza, possibly due to a filter-passing organism; and catarrhal and infectious jaundice.

Angio-cholangitis predominates in tetrachlorethane poisoning and relapsing fever, liver degeneration in phosphorus and chloroform poisoning and yellow fever, blood destruction in meta-dinitrobenzol poisoning and malaria.

The depth of the jaundice depends on the degree of cholangitis that is present, and is of secondary importance to liver degeneration and blood destruction; hence the mortality is low in catarrhal and infectious jaundice although the jaundice is deep, and high in chloroform poisoning although it is slight.

Treatment.—Preventive treatment is provided against chemical poisoning in industries by strict regulations. Delayed chloroform poisoning occurs chiefly in people with pre-existing acidosis and fatty change in the liver. The urine of patients, especially children, should be tested for acetone and diacetic acid before operation. Carbohydrate starvation is an important cause of acidosis, and dextrose should be given freely during the period of preparation.

Preventive measures against diseases such as malaria, yellow fever, and relapsing fever,

which are carried by insects, have given excellent results. The destruction of rats would lessen the outbreak of spirochætosis icterohæmorrhagica.

Curative treatment consists in giving a specific drug, if one is known, such as quinine in malaria and arsenobenzol compounds in relapsing fever and syphilis.

Apart from prevention and specifics, there are certain lines of treatment valuable in all forms of toxæmic jaundice. The patient must be kept in bed and given a light diet with reduced fat and protein. Morning salines are useful to get rid of toxins, but calomel, which is likely to cause mercurial stomatitis, and violent purges should be avoided. Jalap, rhubarb, and senna require the presence of bile to produce their full action and should never be administered in jaundice. If salines are insufficient, aloes and colocynth are good aperients. Sodium bicarbonate and citrate should be given, but chloroform water is a bad vehicle owing to its poisonous action on the liver.

In severe cases nothing but fluids are allowable.

If vomiting is troublesome, whey and citrated milk are best. Fluids, including imperial drink (lemon syrup 1½ oz., acid tartrate of potassium 1 dr., water to 20 oz.), should be unrestricted.

A valuable mixture is:

℞	Sod. cit. ʒi.
	Pot. cit. ʒss.
	Sod. bicarb. ʒss.
	Caffein. cit. gr. iii.
	Syr. aurant. ʒi.
	Aq. ad ʒi.
	T.d.s.

The worst cases require sodium citrate and sodium bicarbonate in ½-dr. doses every two or three hours, and glucose 4 dr. in 10 oz. of water should be given every eight hours by the rectum.

If symptoms of icterus gravis (see LIVER, ACUTE YELLOW ATROPHY OR), such as headache, severe vomiting, drowsiness and delirium, appear, immediate recourse must be had to intravenous salines containing 2 dr. of sodium bicarbonate to each pint, twice a day, until they subside.

In suppression of urine, dry cupping over the lumbar region often starts diuresis.

If the patient recovers from icterus gravis, cirrhosis of the liver may supervene. In such cases, if there is marked ascites more

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than one tapping may be necessary, and the following mixture is recommended :

R̄ Sod. sulph. ʒss.
Pot. cit. ʒss.
Sod. bicarb. gr. xx.
Syr. aurant. ʒi.
Aq. ad ʒi.
T.d.s.

In some of these cases regeneration of liver tissue proceeds and signs of cirrhosis disappear, but the liver is damaged, and care must be taken not to overwork it. Alcohol and irritating articles of food must be given up, and the diet must be light as in ordinary portal cirrhosis.

SIMPLE OBSTRUCTIVE JAUNDICE

Etiology.—This variety of jaundice is caused by mechanical obstruction of the large bile-ducts, due to some local cause. The causes may be classified as follows :

1. Obstruction of the lumen.

- (a) Inflammatory : Cholangitis, simple, suppurative, or obliterative.
- (b) Mechanical : Calculi, worms, ruptured hydatid cyst, new growths of the bile-duct.

2. Pressure from without.

- (a) Tumours in the liver, such as new growth, gumma, and hydatid cyst.
- (b) Enlarged glands at the hilum, malignant, syphilitic, tuberculous, or lymphadenomatous.
- (c) Malignant disease of the pancreas ; acute or chronic pancreatitis.
- (d) Abdominal tumours, such as malignant growths of the duodenum or stomach ; renal or ovarian tumours ; abdominal aneurysm.

3. Cicatricial contraction.

Gumma and duodenal ulcer.

4. Spasm of duct muscle.

Emotional jaundice.

Symptomatology.—Hepatogenous or obstructive jaundice shows some definite differences from toxæmic jaundice.

In the latter condition the jaundice is of little importance compared with the blood destruction, liver degeneration, and general toxæmia ; in the former it may be the outstanding feature.

Obstructive jaundice is more complete and more lasting. It is only in this form of the disease that the skin attains the deep-green colour known as black jaundice, and becomes the seat of xanthoma and telangiectases. In

infants the newly erupted teeth may be green.

The stools are white in the more complete cases of obstruction and may contain no stercobilin, whereas in toxæmic jaundice they are often coloured, and when white are never free from stercobilin. Their clay-coloured appearance is partly due to fat in a finely divided condition. Although their formation is inhibited after a few days, bile-salts are present in greater amount and for a longer period than in toxæmic jaundice, and their presence in the blood causes bradycardia.

The systemic blood-pressure is lowered, the coagulation-time of the blood is three to four times longer than normal, and the red blood-cells are larger and more resistant to hæmolysis in hypotonic saline, whereas in toxæmic jaundice they are usually smaller and more fragile. Nine times as much bile pigment may be present in the blood-serum in obstructive as in toxæmic jaundice.

Absence of bile-salts in the intestine interferes with the digestion of fats and proteins, but the metabolism of sugar, fat, and protein in the liver cells is not affected. The bile, though not antiseptic, is antitoxic, and its absence from the intestine allows the absorption of toxins. The presence of bile-salts in the blood leads first to headache, irritability, and depression, and eventually to degeneration of the hepatic cells and interference with their metabolic functions.

Products of the incomplete catabolism of proteins cause auto-intoxication, with capillary oozing and cerebral symptoms such as convulsions and coma. This is the danger of prolonged obstructive jaundice.

CATARRHAL JAUNDICE

Catarrhal jaundice is probably a mild form of toxæmic jaundice.

Etiology.—There is much to be said in favour of regarding catarrhal jaundice as a specific infective disease, causing a transient hæmic infection with angio-cholangitis and mild hepatitis. The disease is chiefly sporadic, but small epidemics are not uncommon.

Both catarrhal and infective forms agree in showing a definite and parallel seasonal and age-incidence, and are much commoner locally or generally in some years than in others. Children and young adults are most susceptible ; 42 per cent. of all cases occur in children under 10. Infants and old people are seldom attacked. The disease is very

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common, and is most prevalent in the winter months.

The incubation period is about four days, and there is strong evidence that the infection is air-borne. The specific organism is unknown. The geographical distribution is very wide; the disease is commonest in Europe, North America, and Australia, rarest in the tropics.

Pathology.—Owing to its benign nature the pathology is very imperfectly known.

Symptomatology.—There is often a prodromal period of about a week's duration, but this may be absent. The prodromata consist of headache, nausea or vomiting, anorexia, malaise, and aching in the limbs and neck muscles. Constipation is much commoner than diarrhoea. At the end of this period jaundice nearly always occurs, but, in epidemics, cases without jaundice have been recorded. The jaundice may be slight and transient, or intense and long continued. Pruritus is very rare in children, but troublesome in adults. With the onset of jaundice there is often great irritability and depression of spirits. The liver is a little enlarged and tender, and there is dull aching in the right hypochondrium. The spleen is seldom enlarged in sporadic cases.

Bile is present in the urine, and is often noticed before the skin becomes icteric. The urine is often scanty. Albuminuria is very common, but casts are seldom present. Well-marked cases have a slight urinary crisis with a feeling of well-being but without diminution of jaundice, which is suggestive of the termination of an infection. The stools are putty-coloured, but always contain stercobilin. The pulse-rate is raised in children throughout the illness; in adults it often falls to about 40 with the onset of jaundice.

Some cases are febrile when jaundice has appeared, and probably the majority are so in the prodromal period. A temperature of 100°-101° F. is common in children, and it may range from 102°-104° F. in severe cases.

When the disease is mild, especially in children, there is little interference with the general health, but when severe there are considerable wasting and weakness with slow convalescence.

Xanthopsia is rare at all ages.

Relapses are seldom seen. Long-continued cases with varying jaundice are probably due to secondary infection of the bile-ducts. Sub-acute or acute yellow atrophy is a very rare sequel. The subacute cases may recover and develop cirrhosis of the liver.

Diagnosis is usually easy in children and young people. Jaundice with slight constitutional symptoms is almost always due to this common disease. Severe cases may resemble influenza followed by jaundice; but jaundice is rare in most epidemics of influenza, and herpes and pulmonary signs are common in cases of influenza sufficiently serious to cause jaundice. The greater severity of the symptoms; and the presence of other physical signs differentiate pyæmia, pneumonia, enteric fever, and other diseases associated with toxæmic jaundice from this disease.

In the middle-aged, diagnosis is more difficult. Jaundice with few or no other symptoms may be the first sign of *malignant disease of the pancreas or liver*, the presence of which is suspected only when the jaundice persists and deepens. Catarrhal jaundice can be diagnosed from *cholangitis* by the periodic attacks of pain and fever with deeper jaundice in this disease.

Prognosis.—With the rarest exceptions the patient recovers completely. In children and young adults a good prognosis can be given, but it must be more guarded in the case of the pregnant and middle-aged.

Treatment.—The patient should be confined to bed; mild cases require little alteration in diet. Cases with severe vomiting must have milk, citrated milk, or hot water, according to the degree of tolerance met with. When jaundice is established, fats and proteins should be restricted, but fish, chicken, eggs, broth, toast, and milk puddings are allowable. The patient's own appetite is often a good guide.

Salines are valuable, and if an additional aperient is required, aloes can be given. A good mixture is:

Ry Sod. bicarb. gr. x.
Inf. rhei ʒi.
Inf. gent. co. ad ʒi.
T.d.s.

To this mixture tinct. nucis vom. 7½ min. can be added in convalescence.

For pruritus, sponging with carbolio acid 1 in 40, or alkaline baths, often give relief. If not, pilocarpine ½ to ⅓ gr. subcutaneously, or calcium lactate 10 gr. by the mouth three times a day, can be given.

EPIDEMIC JAUNDICE

Apart from the epidemic jaundice of infants, probably three distinct diseases come under this heading, namely: (1) Epidemic

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- Catarrhal Jaundice, (2) Infectious Jaundice, (3) Spirochaetosis Ictero-haemorrhagica.

(1) EPIDEMIC CATARRHAL JAUNDICE

Ordinary catarrhal jaundice is met with not uncommonly in epidemic form. It may be quite mild, like the sporadic form, or more severe. Prodromal symptoms, albuminuria, and splenic enlargement are more often met with. It is air-borne, has an incubation period of about four days, and usually affects the younger children in schools, though classes of older children are by no means exempt. Poor and rich alike suffer. Small epidemics are common in England and Scotland, as well as in the rest of its wide geographical range, and usually occur in the winter months. The diagnosis, prognosis, and treatment are the same as in the sporadic form.

(2) INFECTIOUS JAUNDICE

This form of jaundice is toxæmic, but is sometimes called epidemic catarrhal jaundice.

Etiology.—The disease was common in Gallipoli and Mesopotamia during the War, and perhaps the epidemics of jaundice in the South African War and in the American Civil War were of this nature. The specific organism is unknown, but the *Spirochæta icterohaemorrhagica* has been proved to be absent. The infection is probably water-borne and not spread directly from man to man. It was most prevalent from October to January during the War, and the incidence was the same as that of diarrhoea and dysentery.

Pathology.—This is imperfectly known, but in a patient who died in convalescence there were degeneration of the liver-cells in the centre of the lobules, some regeneration of hepatic cells with newly formed bile-ducts, round-celled infiltration and young connective tissue in the portal spaces. The bile-ducts were patent. The primary condition is probably a hepatitis, and not an ascending cholangitis.

Symptomatology.—There is a prodromal period with anorexia, nausea, occasional vomiting, and diarrhoea or constipation. Later, fever sets in with chilliness or shivering, headache, and vomiting, and there may be pains all over. Four days after the beginning of the pyrexial period, jaundice appears accompanied by white stools and bile-stained urine, and lasts seven to ten days. The liver and spleen are both enlarged, and the liver is tender. Albuminuria is common, but casts are usually absent from the urine. There is nearly always

increased cardiac dullness to the right of the sternum, and the pulse is quickened. There is much loss of flesh, and convalescence is slow.

Icterus gravis is an uncommon complication which occurs about ten days after the appearance of jaundice.

Diagnosis.—The disease differs from true epidemic catarrhal jaundice in the more severe constitutional symptoms, the more regular occurrence of a prodromal period, the invariable enlargement of the spleen, and the frequency of dilatation of the right side of the heart. It differs from *spirochaetosis icterohaemorrhagica* in the absence of herpes, hæmorrhages, or severe toxæmia. In *spirochaetosis* the spleen is not enlarged, but the lymphatic glands are swollen, the jaundice is often slight so that the stools remain coloured, and in many cases there is a secondary rise of temperature. In epidemic jaundice, examination of the blood and urine for *spirochaetes* is negative.

Prognosis.—The outlook is usually good, the death-rate being only 0.3 to 0.4 per cent. Symptoms of *icterus gravis* make it serious.

Treatment.—Rest in bed, light diet and unlimited fluids are necessary, and orange or lemon juice is advisable. Salines and a mixture containing sodium bicarbonate and potassium citrate are the best medicinal treatment. Calomel should not be given. *Icterus gravis* requires the treatment mentioned under toxæmic jaundice.

(3) SPIROCHAETOSIS ICTERO-HAEMORRHAGICA

A general infection with special incidence on the liver and kidneys caused by the *Spirochæta icterohaemorrhagica*. It is almost synonymous with Weil's disease.

Etiology.—The spirochæte has an average length of 8 or 9 μ . Its ends are sharp, and it has two or three large or four or five smaller waves. It can be cultivated anaerobically. Rats act as a reservoir of the disease, and man is generally infected by food or drink contaminated by their urine. The spirochæte can also penetrate the unbroken skin. Wet trenches during the War and wet parts of coal mines in Japan have been especially associated with outbreaks. Butchers' shops and slaughter-houses are also centres of infection. The organism dies in urine and faeces in twenty-four hours and in contaminated soil in three days.

The disease occurs almost all over Europe and in Asia, Africa, and America, and is very prevalent in the Mediterranean area. The spirochæte has been found in rats in London

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and elsewhere in England. Young adults are most prone to the infection, but children and old people are not exempt.

Pathology.—The mucous membrane of the ampulla of Vater, with the duodenum around it, is plum-coloured and oedematous, and there is some inflammation of the rest of the duodenum. The common bile-duct is normal. The liver is swollen, and infiltrated with polymorphonuclear leucocytes. The changes in its cells vary from fatty degeneration to extensive necrosis, and there is angio-cholangitis. Spirochætes are present throughout its substance. Subacute yellow atrophy may be a sequel. The kidneys in mild cases show cloudy swelling, in severe ones necrosis with hæmorrhages. Small hæmorrhages may be found under the pericardium, pleuræ, and peritoneum, and large ones in the lungs. Jaundice is due to angio-cholangitis and obstruction at the ampulla of Vater.

Symptomatology.—The incubation period is six to eight days. The onset is sudden, with shivering, headache, pains all over the body, diarrhœa, and prostration. The temperature rises to 102° F. or more, lasts ten to fourteen days, and falls by lysis, but there is fairly often a secondary rise in the third week. The conjunctivæ are injected, and herpes, usually hæmorrhagic, appears in 45 per cent. of the cases. The pulse-rate is slow for the degree of pyrexia, averaging 75 to 85. Hæmorrhages occur in severe cases from the nose, ears, gums, stomach, and bowel and under the skin, before the appearance of jaundice. Jaundice develops between the fourth and seventh days, reaching its maximum intensity on the tenth to twelfth. In Flanders only 60 per cent. of the cases showed jaundice. In some epidemics the jaundice is very light, in others deep.

Constipation with pale brown stools, and bilious urine, are usual in jaundiced cases. The liver is enlarged and tender, but the spleen is very seldom palpable. Early in the disease there is albumin with hyalin, granular and epithelial casts in the urine, which is scanty and may be suppressed. In favourable cases there is a urinary crisis with polyuria and excessive excretion of urea on the seventh to ninth day of the disease.

General glandular enlargement is present. Bronchitis is common in severe cases. There is great weakness with loss of flesh, and in non-fatal cases recovery is slow.

Parotitis and superficial abscesses are uncommon complications. Remote sequelæ are

irido-cyclitis, hypopyon, and vitreous opacities. Death may be due to toxæmia or icterus gravis.

Diagnosis is difficult before the appearance of jaundice. The disease may be mistaken for *influenzal pneumonia* owing to the hæmoptysis, herpes, injection of the conjunctivæ, and fever, but spirochætes are present in the peripheral blood up to the fifth day, and may be detected under dark-ground illumination or by inoculation of blood into a guinea-pig.

Two drops of 2-per-cent. solution of dimethyl-para-amino-benzene aldehyde in 5-per-cent. HCl added to 5 c.c. of urine give a deep-red colour with excess of urobilinogen and show involvement of the hepatic cells. Cases with cerebral symptoms and petechiæ have been mistaken for *meningococcal meningitis*, but lumbar puncture will prove the presence or absence of this disease. Blood-culture will differentiate it from the *enteric group*. It may resemble severe *trench fever*, but albuminuria with urinary casts and vomiting is rare in that disease.

When jaundice has developed it may be mistaken for *infectious jaundice*, but the presence of hæmorrhages, especially hæmoptysis, of herpes and enlarged glands, and the absence of enlargement of the spleen, are good distinguishing features. Casts in the urine are rare in infectious jaundice. The severity of the toxæmia distinguishes it from the catarrhal jaundice. From *relapsing fever* it can be distinguished by the longer and more wavy spirochæte in the blood in that disease. From all forms of *toxæmic jaundice* it can be differentiated by the presence of spirochætes in the urine, chiefly between the ninth and fifteenth days, while the serum of convalescents after the third week contains a specific agglutination which clumps spirochætes.

Prognosis.—The death-rate is variable. In Flanders it was under 6 per cent., but usually it is 10-20, and occasionally rises to 60 per cent. Serum treatment reduces the mortality considerably. Severe toxæmia and recurrent hæmorrhage are serious signs, and symptoms of icterus gravis make the prognosis bad.

Treatment.—Bed and good nursing are necessary. The diet must be fluid and consist chiefly of citrated milk and whey. Fruit juice must be given, and fluids, including imperial drink, must be unrestricted. Salines and a mixture containing sodium bicarbonate and citrate, as recommended in toxæmic jaundice, should be given. Salvarsan and quinine are useless. Immune horse-serum kills all the spirochætes.

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and is very valuable before jaundice has appeared, but its action is uncertain later. It can be given in three doses of 20 c.c. each on successive days, or 40 c.c. the first and 20 c.c. the second day. It acts better given intravenously than subcutaneously. Severe cases require rectal injections and dextrose 6 per cent. in a pint of saline once or twice a day.

On the appearance of symptoms of icterus gravis, intravenous salines containing sodium bicarbonate are required at once. For heart failure, adrenalin 5 min. four-hourly is advised. For scanty or suppressed urine, cupping over the loins is indicated.

EPIDEMIC JAUNDICE OF INFANTS

This disease, also known as Winckel's Disease, or Epidemic Hæmoglobinuria, is a septicæmia of intestinal origin.

Etiology.—The organism is under dispute. The disease is uncommon in England. Babies about four days old are affected, and it generally occurs in institutions.

Pathology.—The liver is enlarged and, in common with the heart and kidneys, shows fatty degeneration. There are small hæmorrhages in nearly all the organs, under the serous membranes, and in the mucosa of the stomach and intestines.

Symptomatology.—The onset is rapid. The infant is restless, refuses the breast or bottle, and sometimes has a convulsion. Vomiting and slight diarrhoea with pale green alkaline stools are present. The characteristic signs are jaundice and varying cyanosis, which give a curious bronzed appearance. The urine is brownish and contains hæmoglobin, red blood-cells, and casts. Pyrexia, wasting, drowsiness, and leucocytosis are also present. The disease runs its course in three to twelve days. Signs of improvement are increasing diarrhoea with deeper-green acid stools and diminished fever and drowsiness.

Diagnosis.—The cyanosis and hæmoglobinuria distinguish this from all other diseases. From *Bühl's disease*, due to umbilical sepsis, it is distinguishable by the absence of supuration at the navel, and lower fever. In that disease also severe hæmorrhages are generally present, and there may be pleurisy, peritonitis, or meningitis.

Prognosis.—The average mortality is about 30 per cent. Marked cyanosis, high fever, great drowsiness or coma, are very unfavourable signs. More frequent stools of deeper-green colour and acid reaction are favourable.

Treatment.—The infant should be given albumen-water or whey, and a dose of castor oil should be followed by sodium bicarbonate 10 gr. every two or three hours.

Subcutaneous or intraperitoneal salines should be injected twice a day.

HEREDITARY AND FAMILIAL JAUNDICE

The following conditions may be hereditary or familial, viz.: (1) Hæmolytic or Acholuric Jaundice, (2) Simple Family Chlæmia, (3) Hypertrophic Biliary Cirrhosis (see p. 215), (4) Grave Familial Jaundice, (5) Congenital Stenosis of Bile-ducts (see BILE-DUCTS, CONGENITAL STENOSIS OF).

HÆMOLYTIC JAUNDICE

This disease is also known as Acholuric Jaundice, Chronic Simple Jaundice with Splenomegaly, Familial Splenomegalic Jaundice, Hæmolytic Jaundice without Splenomegaly.

Etiology.—The disease is thought to be due either to the formation of red blood-cells of undue fragility, or to a defect of the spleen, which instead of destroying worn-out corpuscles makes normal corpuscles fragile and then destroys them. Syphilis plays no part in the etiology.

The disease may be sporadic, familial, or hereditary, and may be congenital or appear in childhood. The sexes are equally affected, and transmission is by direct descent. Isolated cases sometimes occur in which the disease is acquired in adult life; in these the symptoms are, as a rule, more severe.

Pathology.—The liver is large and firm, but the cells are normal except for the presence of hæmosiderin. There is no angio-cholangitis, and the bile is quite fluid. The spleen is nearly always very large and hard, shows true hyperplasia, and contains much hæmosiderin; the kidneys also contain this pigment.

Symptomatology.—The skin and conjunctiva show a slight icteric tinge. The urine is highly coloured and contains excess of urobilin, but no bile pigment, though the latter is present in the blood-serum. The stools are well coloured. The spleen is usually very large and hard and may extend below the umbilicus, but in a few families there is no splenic enlargement. The liver is large, smooth, and firm. Anæmia is nearly always present. In an average case the red cells number 3 or 4 millions per c.mm., but they may fall to less than 2 millions.

The red cells are fragile, and hæmolysis takes

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place in saline of between 0.6 and 0.42 per cent. instead of between 0.42-0.3 per cent. They are small, and many contain basophil granules. Nucleated red cells are rare except in acquired cases. Slight exacerbations are frequent after over-exertion or exposure to cold. Severe exacerbations are not uncommon in some cases, and are probably attributable to cholangitis. In these the anaemia becomes greater, the jaundice deeper and accompanied by bile-pigment in the urine and by pale stools, and there is fever. The liver is tender and painful.

Sometimes there are recurrent attacks of acute abdominal pain, probably due to small calculi. In mild cases the general health is good. Clubbing of the fingers and xanthoma are unknown. In acquired cases the general health is more apt to suffer, and there may be anaemia of pernicious type.

Diagnosis.—The disease is most often mistaken for *splenic anaemia*, but in this there is no undue fragility of red cells, jaundice is not persistent, and the general health suffers more. Severe acquired cases may be taken for *pernicious anaemia*, but in this disease splenic enlargement is uncommon and never so great as in hæmolytic jaundice. Normoblasts, megakaryoblasts, and poikilocytes are common in pernicious anaemia and the red corpuscles are not fragile, whereas nucleated red cells are relatively uncommon in hæmolytic jaundice.

During an exacerbation the disease resembles *hypertrophic biliary cirrhosis*, but the fragility of the red cells is a safe distinguishing feature.

Prognosis.—Although the health is never so good as in a normal person, the capacity for work is impaired but little, and life may be prolonged to old age in mild cases.

Treatment.—Most cases only require iron compounds from time to time, and rest in bed during pyrexial periods. In bad cases the treatment is splenectomy. After operation wonderful improvement in the general condition takes place—the anaemia disappears, the jaundice is lessened or altogether vanishes—but the red cells generally remain unduly fragile.

If the patient refuses operation, a prolonged course of X-ray treatment to the spleen will cause considerable improvement.

SIMPLE FAMILY CHOLÆMIA

A familial disease characterized in well-marked cases by slight persistent jaundice.

Etiology.—The disease is due to slight angiocholangitis of unknown causation. It

affects both sexes. It is very rare in England, but is commoner in France. It appears to be related to hypertrophic biliary cirrhosis, which may be hereditary but, like this disease, is more often familial.

Symptomatology.—In pronounced cases there is slight persistent jaundice of the conjunctivæ and skin, in others there is xanthoderma or melanoderma and sometimes xanthelasma of the eyelids without jaundice. Bile pigment is present in the serum but not in the urine. Urobilinuria is usual. The liver and spleen may be slightly enlarged or of normal size. The general health is well maintained, but dyspepsia and rheumatic pains are said to be common.

Diagnosis.—The disease can be distinguished from hæmolytic jaundice by the larger liver and spleen and the fragility of the red corpuscles in the latter disease. Urobilinuria and the presence of bile pigment in the serum serve for the recognition of cases without actual jaundice.

Treatment.—Administration of urotropine and salicylates for the angio-cholangitis is the most hopeful line of treatment.

Prognosis.—As regards the duration of life the prognosis is good, but as regards cure it is bad.

GRAVE FAMILIAL JAUNDICE

A toxæmic jaundice which is present at birth or appears soon afterwards, and often attacks several infants in one family, but may occur sporadically.

Etiology.—In most cases the mother is healthy during pregnancy, but in one instance severe vomiting occurred in each pregnancy, and in one or two others the mother became jaundiced at about the sixth month of every pregnancy. The disease is toxæmic, the toxin being derived from the mother's blood. Perhaps different toxins may be the cause in different families. As in toxæmic jaundice due to known chemical or microbial poisons, the chief action, in some cases, is to cause angio-cholangitis, in some destruction of red blood-cells, and in others degeneration of the hepatic cells. There is a close relationship to congenital stenosis of the bile-ducts, which differs chiefly in its less acute character. Syphilis can be excluded as a factor.

Pathology.—The liver and spleen may be normal in size or enlarged. In cases with great blood destruction there is evidence of new blood-formation in the liver, and the spleen

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gives a marked iron reaction. The liver-cells show fatty degeneration or necrosis, and the organ is the seat of round-celled infiltration or even early fibrosis.

In some familial and sporadic cases the basal ganglia and the nuclei in the medulla are deeply bile-stained and their ganglion-cells degenerated. Such cases have been recorded under the name of *kernicterus*.

Symptomatology.—Signs and symptoms vary greatly, and may differ even in members of the same family. Sometimes jaundice is present at birth, at others it appears shortly afterwards. It may be very deep or very light, especially in anæmic cases. The blood may be normal, or there may be profound anæmia with megaloblasts and myelocytes. Leucocytosis is present in some cases. The stools may be dark throughout, or after the passage of normal meconium they may become white. The urine may or may not contain bile pigment. The liver and spleen may be normal in size or enlarged. Drowsiness, coma, and convulsions are common. Hæmorrhages seldom occur. Some infants die very rapidly, some survive for weeks, others recover completely. In anæmic cases which recover, the anæmia may last for weeks.

Diagnosis.—The history in familial cases makes diagnosis fairly easy. In the others the severe toxæmia, and the anæmia and white stools when present, separate the disease from *icterus neonatorum*. Absence of umbilical suppurative infection, and of cyanosis and hæmoglobinuria from *Winckel's disease*. *Catarrhal jaundice* is very rare at this age, and much milder. *Syphilis* seldom causes jaundice; other lesions are usually present, and the mother's Wassermann reaction is positive.

Prognosis.—Few cases recover. Profound anæmia and marked toxæmia are bad signs. In cases of *kernicterus* which recover, the patient is left mentally defective with hypotonia and choreiform movements.

Treatment.—Antenatal treatment consists in keeping the mother's bowels open and in giving urotropine with intestinal antiseptics. For the baby, intraperitoneal salines twice a day with sodium citrate and bicarbonate by the mouth are best.

ICTERUS NEONATORUM

This benign form of jaundice appears soon after birth and is to be regarded as almost physiological.

Etiology.—Great diversity of opinion exists as to the cause. It is said that in a normal infant bile is scanty or absent in the first twenty-four hours, and secretion is gradually established in the first week, whereas in a jaundiced infant there is excessive secretion of bile in the first twenty-four hours, and some of it is absorbed by the blood and produces jaundice.

Another view is that a flood of toxins reaches the liver when bacteria begin to grow in the previously sterile intestine. The liver at first is unable to destroy them completely, and slight angio-cholangitis is caused. The condition appears in 50–80 per cent. of all children, and the strong and healthy are affected as often as the weak and premature.

Pathology.—The body fluids and all the tissues of the body are bile-stained, including the brain and especially the basal ganglia, which escape in jaundice in adults. The liver is normal, the bile-ducts are patent.

Diagnosis.—At first no certain diagnosis can be made, but this is infinitely the commonest form of jaundice during the first few days of life. If the jaundice deepens after the first two or three days and there are constitutional symptoms, a more serious condition is probably present. *Catarrhal jaundice* is very rare at this age. The history, the toxæmia, and the nervous symptoms distinguish *grave familial jaundice*. In *congenital stenosis of the bile-ducts* deepening jaundice, white stools, and bilious urine are the chief points of difference, and there may be hæmorrhages. In *umbilical infection* there are constitutional symptoms and signs of local sepsis. In *Winckel's disease* fever, cyanosis, and hæmoglobinuria with symptoms of serious illness make differential diagnosis easy. Jaundice is of uncommon occurrence in congenital syphilis; the liver and spleen are enlarged, and other signs of the disease may be present.

Prognosis.—There may be temporary loss of weight, but perfect recovery is the rule.

Treatment.—There is no need for treatment, unless the jaundice persists beyond the usual time, in which case hydrarg. c. cret. $\frac{1}{4}$ gr. can be given two or three times a day.

E. A. COCKAYNE.

JAW, NECROSIS OF (see ORAL SEPSIS).

JOINTS, INFLAMMATION OF (see ARTHRITIS; RHEUMATOID ARTHRITIS; GONORRHEA; SYNOVITIS).

KALA-AZAR

KALA-AZAR.—A protozoal disease evidenced by fever—which runs a very prolonged course of from many months to several years—great enlargement of the spleen and liver, progressive emaciation, and extreme leucopenia; and usually terminating in a fatal secondary bacterial infection.

History and geographical distribution.

—Kala-azar (Black Fever) was first described in Assam, where it spread in an epidemic form over several hundred miles of the Brahmaputra valley between 1881 and 1900, and largely depopulated extensive areas. After the discovery of the parasite, the *Leishmania donovani*, in 1903, what had previously always been looked upon as "malarial cachexia" in many parts of India was found to be a sporadic form of kala-azar. The disease has for the most part lost its tendency to spread in an epidemic form in Assam, except for a recent extension in the Sibsagar district, measures having been taken with considerable success to check its progress in accordance with the writer's recommendations.

On the other hand, the sporadic form has been found to be widely prevalent in the Bengal, Bihar, and Madras provinces of India; whilst a disease which is similar and believed by many to be identical is found in the northern parts of Africa, and to a less extent on the northern shores of the Mediterranean.

Etiology.—In 1903 Leishman described as degenerate trypanosomes certain bodies which he found post mortem in the spleen of a soldier invalidated from India for "malarial cachexia," and immediately afterwards Donovan reported that he had independently discovered similar bodies in Madras, and had obtained them by spleen puncture during life. Bentley and Rogers found the same bodies in the epidemic kala-azar of Assam, and the latter shortly afterwards obtained cultures of the parasite in sterile citrated blood kept at a temperature below 75° F., and traced the development of the flagellate stage.

All ages and both sexes are attacked, but the disease is commoner in children, especially in the African form, which has been called "infantile splenomegaly." It is largely a house or site infection, one person after another in a family being attacked. Complete removal of the healthy persons of a community to a new site only a few hundred yards from the infected

houses suffices to prevent the spread of the disease—a fact which excludes a flying insect as the carrier of the infection. Dodds Price has eradicated kala-azar from the labour forces of a number of Assam tea gardens by carrying out such segregation measures. As a result of his culture work the writer suggested the bed-bug as the most likely infective agent, and Patton subsequently obtained development of the parasite up to the flagellate stage in bed-bugs fed on patients showing the parasite in their



Fig. 42.—Kala-azar parasites from the spleen, stained.

peripheral blood, though not frequently enough to establish this as the ordinary mode of infection. This problem is not yet fully solved.

Pathology.—The parasite (Fig. 42), as seen in the spleen-puncture films, is a minute oval body, about the size of a blood-plate, with a larger rounded nucleus and a smaller rod-shaped one.

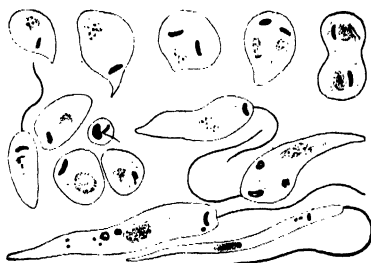


Fig. 43.—Kala-azar parasites in culture. (After Leishman.)

Similar but smaller bodies are found post mortem in the spleen, liver, and bone-marrow, chiefly in the endothelial cells and macrophages. In cultures the organisms increase rapidly in size (Fig. 43), and at the end of two or three days a flagellum develops from the micro-nucleus, now situated at the anterior end of the greatly elongated and actively motile organism. Development can also be obtained on neutral blood-agar, but it is inhibited if the medium be markedly alkaline.

After death the *spleen* is nearly always greatly enlarged, commonly weighing 3 lb., and the *liver* is increased in a lesser degree. The *blood* shows a characteristic leucopenia, with disproportionate reduction of the polymorphonuclear cells, so that the number of these cells usually falls from 6,000 to about 1,000, and not rarely to 500 or even 250 per c.mm. In uncomplicated cases the ratio of the white to the red corpuscles, in all but the earliest stages, is less than 1 to 1,500, which practically distinguishes the disease from chronic malaria and other conditions which could well be confused with it. This loss of the majority of the polynuclears is also of great pathological importance, as it strongly predisposes to secondary bacterial infections, such as cancrum oris, septic infections, pneumonia, dysentery, plithisis, etc., to one of which eventually the patient usually succumbs. The parasites have been cultivated from the peripheral blood, in which they are sometimes also found in small numbers within polymorphonuclear leucocytes.

Symptomatology.—Kala-azar begins with fever, usually at first of a remittent type, and often showing two remissions of several degrees within twenty-four hours. This is of considerable diagnostic value, especially with respect to typhoid fever, for which kala-azar is often mistaken in its early stage. After two or more weeks the temperature declines to a low intermittent or continued form, only to rise again at irregular intervals. A low continued temperature of about 99°–101° F. is commoner in kala-azar than in other tropical fevers. The fever persists for months and not rarely for over a year, but in very chronic cases the temperature may become normal for a time and again relapse. After the first few weeks the general symptoms are very slight, even with a considerable degree of fever, although the pulse tends to remain rapid. The patient may have a very good appetite and make light of his condition. Emaciation is rapid, and in dark races the face has a dusky or earthy hue. The spleen early becomes greatly enlarged in most cases, and may reach the navel within a short time from the onset of the fever. Later it may extend to the right anterior superior spine of the ilium. Dodds Price, however, has recorded cases with very little splenic enlargement. The liver is not usually much enlarged for the first six months or so, but may eventually extend to the navel. Anæmia is constant, but is comparatively slight, over

2,500,000 red corpuscles per c.mm. being usually present, while the white corpuscles are disproportionately reduced, as already mentioned. The colour index is about normal. Death rarely results directly from the disease itself, but from some complicating bacterial infection favoured by the deficient phagocytosis. If a septic infection or pneumonia be followed by leucocytosis, which is rarely the case, the patient may eventually be completely cured of the original disease, the spleen ultimately disappearing beneath the ribs.

Diagnosis.—With some experience it is usually possible to recognize an advanced stage of the disease from the great emaciation, enlargement of the spleen and liver, and peculiar earthy colour, generally unaccompanied by evident anæmia. In chronic malaria with splenomegaly the emaciation is usually much less and the anæmia greater. The differentiation from malaria can nearly always be made by giving 30 gr. of quinine daily for a week, which will stop malarial paroxysms for a time at least, but will not materially affect the fever of kala-azar. The diagnosis is far more difficult in the early stage of kala-azar, when it is often necessary to look for the parasite either in the peripheral blood or by spleen or liver puncture. The former is troublesome and uncertain, while the latter is not without danger, especially in very anæmic cases. It is safer to puncture the liver than the spleen, but the parasites are less readily found in the former, and it may not be enlarged. By giving calcium chloride beforehand, and afterwards applying a bandage and keeping the patient in bed for a day, puncture of the spleen with a fine needle is generally safe in experienced hands, except in very anæmic cases. Safety can be ensured by first estimating the coagulability of the blood with Wright's instrument, and only doing spleen puncture if the time does not exceed five minutes.

Prognosis.—In the epidemic form this was nearly hopeless. Recovery followed in only 4–10 per cent. of Dodds Price's cases in Assam tea gardens, but when it occurred it was complete and permanent. In the sporadic form a somewhat larger proportion of cases recovered, not rarely after some leucocyte-increasing bacterial infection, such as cancrum oris or other septic condition.

Great leucopenia and anæmia are grave signs. The degree of enlargement of the spleen is of no prognostic value. Steady loss of weight is most serious, and continuous gain is the

most favourable sign, especially if accompanied by increase of the polymorphonuclears.

The introduction of treatment by antimony salts intravenously, as described below, has completely altered the prognosis of kala-azar, for the case-mortality in Assam has already fallen to below 25 per cent., while it is lower still in sporadic cases treated in hospital before the disease has reached a very advanced stage.

Treatment.—Until recently we had no reliable treatment of kala-azar. Careful nursing and measures to raise the number of polymorphonuclears, such as the subcutaneous injection of staphylococcus vaccines by the writer or sterile turpentine by Muir, resulted in the recovery of some 25 per cent. of sporadic cases.

The discovery of the flagellate stage of the protozoal parasite of kala-azar revealed the close affinities of this disease with sleeping sickness, which in its turn led to the use in the treatment of kala-azar of drugs which had been proved experimentally to be inimical to the trypanosome of sleeping sickness, but with little success until the soluble antimony salts were given intravenously. Di Cristì and Caronia first proved and recorded the success of tartar emetic intravenously in curing the infantile or Mediterranean form of kala-azar in Sicily, and the writer independently obtained a similar success a few months later in the Indian form in Calcutta, and subsequently demonstrated that the corresponding sodium salt was slightly less toxic than, and consequently preferable to, potassium antimony tartrate (tartar emetic). Later he also obtained similar curative results with a practically non-toxic colloid, antimony sulphide. All these preparations must be given intravenously. Antimony oxide dissolved in glycerin has not proved effective in the writer's hands.

The potassium and sodium antimony tartrates have to be pushed cautiously up to full doses and given twice a week for several months in order to cure the disease. A 1- or 2-per-cent. solution may be used, beginning with 2 or 3 c.c. of the 2-per-cent. solution in adults, and increasing by $\frac{1}{2}$ c.c. at each dose up to 5 c.c. or a little over, so long as no toxic symptoms are produced except very temporary nausea, but if actual sickness occurs the dose should be reduced for a time. Under this treatment the fever usually ceases in about three weeks, but it is essential to success to continue the injections for three months or more to prevent a relapse, which may prove

resistant to the drug. After the temperature has fallen to normal, a slight rise will follow an injection for several weeks, indicating the liberation of toxins from destroyed parasites. There is no absolute guide as to when a complete cure has been effected, but disappearance of the spleen beneath the costal margin, with recovery of all lost weight and an increase of the leucocytes to above the normal number are the safest indications for stopping the treatment. At Kulna, in Bengal, this treatment has been so successful in Muir's hands that the disease has nearly disappeared from the neighbourhood of his hospital. With a vigorous campaign sporadic kala-azar should be stamped out of the sporadic areas, and the new treatment is also proving of great value in checking the spread of the disease in Assam.

LEONARD ROGERS.

KATATONIA (see CATATONIA).

KELOID (see CHELOID).

KERATITIS (see CORNEA, AFFECTIONS OF).

KERATODERMIA (see HYPERKERATOSIS).

KERATODERMIA BLENORRHOICA
(see GONORRHOEA).

KERATOSIS PILARIS (see ICHTHYOSIS).

KERION (see RINGWORM).

KERNICTERUS (see Grave Familial Jaundice, under JAUNDICE, p. 148).

KIDNEY, AMYLOID DISEASE OF (see AMYLOID DISEASE).

KIDNEY, CONGENITAL ANOMALIES OF.—The anomalies to which the kidney is liable may be that (1) of number or (2) of position, or may consist in (3) fusion of the two organs, or in (4) the presence of aberrant vessels.

1. Anomalies of number.—Absence of both kidneys has been observed in the fœtus.

Absence or complete congenital atrophy of one kidney, more frequently the left, occurs about once in 2,500 cases (Morris). It is slightly more common among males than in females, and is usually accompanied by some defect in the genital organs. Although the ureter is nearly always absent, its orifice is sometimes present and may lead into a blind pocket. Occasionally the corresponding side of the trigone is undeveloped. The remaining kidney is generally hypertrophied, but appears

KIDNEY, CONGENITAL ANOMALIES OF

to be liable to disease. The importance of this condition lies in the fact that in many cases the single kidney has been removed. The **diagnosis** is made by cystoscopy and catheterization of the ureters.

In a few cases three, four, or even five kidneys have been found.

2. Anomalies of position.—Congenital displacements nearly always occur in a downward direction. The organ cannot be replaced in its normal position, and may lie low down in the loin, in the iliac fossa, on the brim of the pelvis, or in the hollow of the sacrum. It occurs about once in a thousand cases (Morris), but is very seldom extreme. The other kidney may be absent. There may be no **symptoms**, or the misplaced kidney may press on the rectum, uterus, bladder, sacral plexus, etc. It more frequently causes symptoms in females than in males, although it is commoner among the latter. The **diagnosis** is rarely made, for a solid tumour in one of the localities mentioned may not be recognized as a kidney, as it is often misshapen. It is confirmed by ureteric catheterization and pyelography. If the kidney gives rise to serious symptoms it should be removed, provided that its fellow is healthy.

3. Fused kidneys.—The best-known example of this condition is the *horseshoe kidney*, which occurs about once in six hundred cases (Morris). Here the kidneys are connected across the middle line by a band of tissue, which may be merely fibrous but is generally a thick mass of renal tissue derived from the cortices of both. The isthmus lies in front of the great vessels, and is crossed anteriorly by the two ureters. The kidneys lie much closer together and at a lower level than the normal. Supernumerary renal vessels are generally present. This condition causes no symptoms, and only becomes important when one kidney is diseased. In **diagnosis** a horseshoe-shaped tumour may be felt in front of the vertebral column. The diagnosis is confirmed by pyelography, the shadows of the renal pelvis being closer together and lower than normal.

A more complete fusion results in an *irregular lobulated kidney mass*, which generally lies on the promontory of the sacrum or in the true pelvis. The presence of two ureters is the only indication that it is derived from both kidneys. It has been removed with disastrous results.

Occasionally the kidneys are fused from end to end. In this case one crosses the middle line and is united to the lower pole of the other. There are two varieties of this *long kidney*

according as the pelves look in the same or in opposite directions.

4. Aberrant renal vessels are very common, and are found in about 20 per cent. of cases. As a rule they do not give rise to symptoms, or cause any interference with the renal secretion, and are only of importance when the kidney has to be operated upon. Occasionally an aberrant vessel supplying the lower pole of the kidney produces hydronephrosis. This is the most common form of the abnormality, and the vessel may spring either from the aorta or from the renal artery, and enters the kidney below the hilum. In the majority of instances it passes in front of the ureter or pelvis. Kinking of the ureter, with subsequent hydronephrosis, may be caused by an aberrant vessel passing either in front of the ureter, to its insertion in the posterior part of the lower pole of the kidney, or behind it, to its insertion into the anterior aspect of the organ. In these cases the dilatation commences at the point where the vessel crosses the ureter. The symptoms are those of hydronephrosis (q.v.), but the cause of the distension is seldom diagnosed before operation. It may, however, be suspected if no other cause for the condition is found, and if in pyelography the shadow of the ureter shows a distinct kink just below the renal pelvis. **Treatment** consists in dividing the aberrant vessel, but the kidney should not be returned into the wound until some evidence of return of the circulation in the lower pole has been observed. Occasionally either a partial or a complete nephrectomy may be necessary.

Aberrant vessels to the upper pole spring, as a rule, from the renal artery or the aorta, but occasionally they arise from the middle capsular or the inferior phrenic. They give rise to no symptoms.

Aberrant vessels are the rule in misplaced or fused kidneys. In the horseshoe kidney as many as ten may be present, but usually either three, four, or five arteries are found. The isthmus is supplied by one, or occasionally two, short arteries springing from the aorta, close to its termination, while the lateral kidney masses are each supplied by one or more arteries. If more than one is present, the upper artery corresponds to the true renal, and enters the kidney at the hilum; the others are aberrant, and enter below this point. In the fused pelvic kidney three or four arteries are usually found; they spring from the termination of the aorta, the common or internal iliacs, or from the middle sacral. Their distribution to the kidney mass is exceedingly irregular. J. SWIFT JOLY.

KIDNEY, CYSTS OF

KIDNEY, CYSTS OF.—Small multiple retention cysts are frequently found in kidneys affected with chronic interstitial nephritis. For hydatid cysts, see HYDATID DISEASE.

Solitary Cyst of the Kidney (*syn.* Serous Cysts).—In this rare condition a large cyst is found springing, as a rule, from one pole of the kidney. It contains a clear straw-coloured fluid, in which traces of urinary salts are found. The cyst may grow to an enormous size, and is frequently partially subdivided by imperfect septa. The **etiology** is unknown. The **symptoms** are tumour and pain. The situation of the tumour depends on the position of the cyst; it is smooth, rounded, and moves on respiration. If the cyst springs from the upper pole, the kidney is displaced downwards. The pain is generally a dull ache, but acute pain resembling colic may result from pressure on the ureter, or displacement of the kidney. The **diagnosis** is rarely made, and as a rule the condition is only found post mortem. **Treatment** consists in excision.

Polycystic Disease (*syn.* Congenital Cystic Disease; Cystic Degeneration).—Usually two types of this disease are described, the congenital and the late, but the same pathological process underlies both conditions. In the congenital form the kidneys may be enlarged at birth, and their size even complicate parturition, or the enlargement may be noticed afterwards. The child rarely lives more than a few months. In the late form the disease generally appears between the ages of 40 and 50, runs a slow course, and ends fatally. It is bilateral, though it may be more advanced in one kidney than in the other. It may be accompanied by a cystic condition of the liver, spleen, or ovaries. It is slightly more common among females than males, and occasionally has been found in several members of one family.

Pathology.—The kidneys become enormously enlarged, but retain their reniform shape. They are transformed into an immense number of cysts, varying in size from a pin's head to a cherry. These may be flattened from mutual pressure. The disease commences in the cortex near the poles, and when it is advanced no renal tissue can be seen with the naked eye. The cysts are filled with either a clear straw-coloured or a brown fluid, which contains albumin and small quantities of urea and urinary salts. The walls are fibrous, and usually lined with a single layer of epithelial cells, and sometimes large numbers of desquamated epithelial cells are present in

the cysts. The glomeruli and convoluted tubules, occupying the spaces between the cysts, undergo pressure atrophy, and are smaller than normal.

Etiology.—This is uncertain. The condition has been attributed (1) to inflammation, (2) to congenital defect, and (3) to neoplasm.

Symptoms.—The heart is usually hypertrophied and the blood-pressure raised in the earlier stages of the disease, and patients may die of cerebral hæmorrhage before the characteristic symptoms appear. At first the tumour may be unilateral, later on another is almost invariably detected on the opposite side. Each has all the usual characters of a renal tumour. It descends below the ribs, fills out the loin, moves on respiration, is generally painless on palpation, and occasionally irregularities due to the projections of cysts on the surface may be felt. The presence of two enormous kidneys, almost filling the whole abdomen, is characteristic of the late stages. Frequently there is a constant ache in the loins. The changes in the urine are those usually associated with renal failure, but, as the progress of the disease is very slow, they are spread over a period of several years. At first polyuria is present, three, four, or even five times the normal amount being excreted daily. The urine has a very low specific gravity, and often contains a trace of albumin. The total excretion of urinary solids may be unaltered at first, but later is diminished. Tube-casts and blood are often found. Hæmaturia is generally slight and intermittent. Finally, the urine becomes scanty, and anuria may set in. Death is usually due to uræmia. [144]

Treatment.—Surgical interference is generally contraindicated, and the patient should be treated as if for chronic interstitial nephritis.

J. SWIFT JOLY.

KIDNEY, FLOATING OR MOVABLE
(see VISCEROPTOSIS).

KIDNEY, GRANULAR (see NEPHRITIS).

KIDNEY, HYDATID DISEASE OF (see HYDATID DISEASE).

KIDNEY, INFLAMMATION OF (see NEPHRITIS; PERINEPHRITIS; PYELITIS AND PYELONEPHRITIS; PYONEPHROSIS).

KIDNEY, INJURIES TO (see ABDOMINAL INJURIES).

KIDNEY, SYPHILIS OF (see NEPHRITIS).

KIDNEY, TUBERCULOSIS OF

KIDNEY, TUBERCULOSIS OF.—Tuberculous disease of the kidney may occur without any active focus of disease being present in the rest of the body. It may constitute the only focus in the urinary system, or may be secondary to active disease in the generative organs or in the lung. Both kidneys may be attacked in miliary form as part of a general tuberculosis, usually in childhood.

Etiology.—Renal tuberculosis is most frequent in early adult life. Formerly it was held that in renal tuberculosis both kidneys were affected; this conclusion was based upon the observations upon late cases in the post-mortem room, but the more exact methods of diagnosis by means of ureteric catheterization and the results obtained by nephrectomy have shown that the disease is at first unilateral. There is no evidence that injury to a kidney is a predisposing factor.

Infection usually reaches the kidney by the blood-stream (*haematogenous infection*), and in early cases may be localized to one kidney. It was originally thought that infection of the kidney only occurred secondarily to tuberculosis of the bladder, ascending by the ureter (*ascending infection*), this opinion being based upon the early symptoms of increased frequency of micturition and pyuria; but it is now shown by the cystoscope that these symptoms may be present without any visible evidence of vesical infection; where there is vesical tuberculosis as well as renal, the pathological evidence is as much in favour of descending as of ascending infection.

Pathology.—Apart from miliary tuberculosis, the infection usually begins at the apex of a pyramid, gradually proceeding to ulceration and destruction of the adjacent renal tissue towards the base of the pyramid. The tissue around is thickened, forming an irregular cavity lined by caseous material, opening into the renal pelvis by a narrow channel. At the same time groups of greyish tubercles may be deposited in the cortical substance of the kidney. Several pyramids may be attacked, forming localized cavities in the kidney, which present as rounded, softened areas on the surface. The kidney may be enlarged at one pole only, or, where the disease is more extensive, may be enlarged generally. In some cases the kidney tissue is replaced by masses of semi-solid, yellowish, caseous material, shut off from the renal pelvis, whilst in others the localized thickening obliterates the lumen of the ureter and a tuberculous pyonephrosis results. In

these cases there will be no evidence in the urine of tuberculous disease ("closed" type).

The infection in all cases spreads to the pelvis of the kidney, the lining membrane becoming studded with small raised elevations or broken down in part into small ulcers. The actual communication with the softened tuberculous area in the kidney may be small and surrounded by thickened fibrous tissue, the lumen being occasionally blocked by caseous débris. The inflammatory process spreads to the submucous coats of the pelvis, which become thickened and infiltrated, while the fatty tissue of the hilum may be sclerosed and adherent.

The ureter is infected in the same manner. The mucous membrane lining the duct becomes raised by tuberculous processes and the lumen narrowed. Fibrous infiltration thickens the wall of the ureter greatly, so that the canal stands out as a rigid tube the size of an ordinary lead pencil, and may easily be palpated per rectum or per vaginam. The lumen of the ureter may be blocked by caseous material from the kidney.

Symptomatology.—The symptoms of tuberculous disease of the kidney are often very misleading, and may be those of persistent cystitis; in fact, gross destruction of the kidney may be present without any lumbar pain. There may be an aching in the loin in the angle made between the last rib and the outer border of the erector spinæ muscle, and occasionally a more acute attack of colic from the passage of caseous material down the ureter or from partial blockage of the duct. The more prominent symptoms are those associated with cystitis, although a cystoscopic examination may show the absence of vesical infection. There is a gradually progressive increased frequency of micturition, at first during the day only, but later during the night also, together with a scalding in the urethra during micturition and pain in the glans penis or in the vulva at the end of the act.

Changes in the urine.—The urine is pale and of opalescent appearance, depositing a small amount of pus. The specific gravity is low owing to the increased amount of urine passed from the diseased side. Red blood-corpuscles are found under the microscope, but visible hæmaturia is variable. In some cases hæmaturia may be present as an early symptom, or may occur as the result of a local injury or of exertion. The last few drops of urine passed during micturition may be tinged with blood

KIDNEY, TUBERCULOSIS OF

when vesical infection has occurred secondarily to the kidney.

Albuminuria is present in all cases. It may be present before the appearance of blood and pus in the urine; in one case under my care the patient was refused life insurance owing to albuminuria eighteen months before the appearance of pus and blood in the urine and before any increased frequency of micturition was noted. Tubercle bacilli are present in the urine.

Physical examination.—As a rule, the tuberculous kidney is not enlarged unless there is partial or complete hydronephrosis. If the kidney can be felt it may be slightly irregular and tender on pressure. The lower end of the ureter may be easily felt per rectum or per vaginam, and this examination should never be omitted when renal tuberculosis is suspected. In the male the thickened ureter can be felt as a distinct, rounded, tender cord above the prostate, passing upwards and outwards, and in the female it may be felt in the lateral vaginal fornix; in both sexes it may often be rolled between the fingers on bimanual examination.

It must not be forgotten that when one kidney is partially or wholly destroyed by tubercle, the remaining kidney may be enlarged by compensatory hypertrophy, in which case it may be palpable and tender on palpation.

Diagnosis.—In a young adult the symptoms of cystitis coming on insidiously and without any apparent cause, such as venereal disease or catheterization, should make the practitioner suspicious of tuberculous disease. The finding of small quantities of pus, blood, and albumin in a pale, hazy urine should be followed by an examination for tubercle bacilli, which, if found, will make the diagnosis more certain. Aching in the loin, tenderness over the kidney on deep palpation, and the detection of a thickened ureter per rectum, will all point to the side affected. At the same time an examination of the prostate and vesicles per rectum and of the epididymis in the male will determine the existence of genital tuberculosis, to which the urinary infection may be secondary.

Further knowledge is gained of the disease by **cystoscopic examination**. The bladder may be found quite normal, even when vesical symptoms are prominent, but it is more usual to find the small, greyish elevations of submucous deposit of tubercle or active ulceration of the mucous membrane. (PLATE 8, Fig. 4, Vol. I, facing p. 313.) The changes in

the appearance of the ureteric orifice may give valuable information. There may be some oedema of the lips of the orifice from cystitis; or tubercles may be seen in the immediate vicinity; or the edges of the orifice are lost in a small area of tuberculous ulceration. In later cases the orifice is displaced upwards and outwards, and appears at the end of a gutter-shaped depression, from the dragging of the thickened and shortened ureter.

In every case in which operation may be contemplated the urine from each kidney must be obtained separately, preferably by **ureteric catheterization**, as the use of a separator in an inflamed bladder might result in bleeding from manipulation. In those cases in which cystoscopy or rectal examination affords no evidence as to which ureter is diseased, both ureters should be catheterized, but when one is diseased the catheter should be passed only into the presumably normal one, as it is necessary to know that the kidney on this side is not only functionally active but is free from disease. The urine collected should be carefully tested for pus, blood, albumin, and tubercle bacilli, the percentage amount of urea should be estimated, and an estimation of the functional activity of the kidney made by the methylene blue or phloridzin glycosuria test.

Skiaographic examination may show some increased opacity in the renal area. The shadows are due to the areas of caseous degeneration and are less distinct than the shadows of calculus.

A *renal calculus* gives rise to greater renal pain, associated with reno-ureteric colic, than tuberculous disease; colic is unusual with the latter, but may be caused by the passage of caseous debris or bloodclot down the ureter. Renal calculus does not lead to increased frequency of micturition in the same continuous manner, and tubercle bacilli are absent from the urine. A calculus impacted in the lower end of the ureter causes thickening of the duct and increased frequency of micturition, but a cystoscopic and radiographic examination will determine the diagnosis.

Prognosis.—The course of tuberculous disease of a kidney is gradually progressive, the renal tissue being progressively destroyed. There may be periods when the symptoms diminish, but after a short time they return. The spontaneous cure of a tuberculous focus in the kidney must be very exceptional, and although some amelioration of symptoms may occur with treatment under general lines, and

by tuberculin injections, our present knowledge is insufficient to expect permanent benefit by anything short of nephrectomy and ureterectomy. When the operation has been performed, further treatment is necessary for some months, and the prospect of cure is greatly enhanced. Even when vesical tuberculosis is present, or when foci are present in the prostate or vesicles, the removal of the primary disease with efficient subsequent treatment has been followed by the disappearance of the secondary foci.

When septic infection is superadded, the course of the disease is much more rapid. The infection may be the result of urethral instrumentation or by the blood-stream, and may lead to pyonephrosis. When bilateral tuberculosis exists, death takes place from anuria or septic toxæmia.

Treatment.—The treatment to be followed depends to a large extent upon the spread of the disease. By the methods described above, it must first be determined that the other kidney is functionally active and not infected, and any infection of the bladder or of other foci in the body must receive consideration. Thus evidence of active tuberculous lesions in the body, such as spinal caries, plithisis, joint-disease, etc., would form a contraindication to operation, but old healed tuberculous lesions, such as an ankylized joint, may be disregarded. Multiple foci of disease in the testes, prostate, or vesicles, again, would negative operative treatment, but small or early foci in these organs can be treated on general lines after the diseased kidney has been removed. Tuberculosis of the bladder would not in itself contraindicate the removal of the primarily infected kidney, as, under appropriate treatment, extensive vesical infection has been seen to clear up when the primary focus has been eradicated. When the disease has attacked both kidneys, operation cannot be advised.

Operative treatment.—When the disease is proved to be unilateral, and in those cases in which other foci of active disease do not exist, there is no doubt that *complete removal of the infected kidney and ureter* holds out a good prospect of cure.

Partial nephrectomy, or the removal of the diseased portion of a tuberculous kidney, is inadvisable. It is frequently difficult to say, even when the kidney is fully exposed, how extensively the disease has spread, and though a localized focus may be palpable in one pole,

the other may contain small foci which would be left were a partial operation performed.

Nephrotomy may occasionally be necessary to relieve urgent symptoms, or as a preliminary to nephrectomy. In cases in which a septic infection is superadded to the tuberculous, a pyonephrosis may be found and drainage may become advisable to relieve the toxæmia, pyrexia, etc., to be followed by nephrectomy when the general condition improves. Again, when bilateral tuberculous disease is present, nephrotomy may be required as a palliative operation.

General treatment.—In cases in which operative treatment is contraindicated or refused, treatment must proceed on general lines, and it is equally essential that after operation every measure should be taken to increase the patient's natural resistance to tuberculous trouble. Good, nourishing food with plenty of milk, cream, cod-liver oil and malt should be given, and the patient should reside, if possible, in a warm climate. The vesical irritability should be relieved by sandal-wood oil, or belladonna suppositories; no local treatment to the bladder by irrigation or operation is permissible. After operation a thorough course of tuberculin injections, given once a week, should be resorted to, commencing with a dose of 1/10,000 mg. of T.R., and gradually increasing so long as no marked reaction occurs. If nephrectomy has not been performed the injections of tuberculin must be given cautiously, as its administration may be followed by a further infiltration in the ureteric mucous membrane, with consequent aching in the affected side owing to increase of intrapelvic pressure.

R. H. JOCELYN SWAN.

KIDNEY, TUMOURS OF.—**Innocent tumours** of the kidney are rare. They are of very little clinical importance, being usually found on pathological examination. Adenomata, fibromata, and lipomata have been found as small localized tumours in the cortical areas of the kidney, but give rise to no symptoms. A case of angioma of the renal medulla has been recorded by the writer which caused such profuse hæmaturia as to endanger the patient's life, necessitating removal of the kidney.

Malignant tumours of the kidney are more common than the benign form, and may be classified as:—

1. Hypernephroma.
2. Carcinoma.
3. Sarcoma.
4. Embryonic mixed tumours.

KIDNEY, TUMOURS OF

1. The *hypernephromata* form the largest group of growths in the kidney—occurring in about 80 per cent. of all renal tumours, and usually after the age of 45. The pathology of this tumour was first described, in 1883, by Grawitz, who asserted that it arose from the growth of small masses of aberrant adrenal tissue which are found in the subcapsular portion of the kidney and have become included in the course of development. This view arose from the histological resemblance of a section of the tumour to that of the suprarenal gland, but more recent work by Stoerk and Shaw Dunn goes to show that these tumours arise in the renal epithelium and must be looked upon as carcinomata. While uncertainty remains as to the origin of the tumours, it is as well to adopt the name of hypernephroma suggested by Lubarsch.

Hypernephromata occur as rounded tumours, usually forming a prominence from the kidney, covered by the capsule and a thin layer of compressed renal tissue. They also spread towards the pelvis, which they ultimately invade, and may gain entrance to the renal vein or even the inferior vena cava. On section a hypernephroma may be found to vary considerably; it may be of firm consistence, but frequently shows areas of necrosis and softening and commonly some dark areas into which hæmorrhage has occurred. The marked feature which is present in almost all cases is the presence of small areas of fairly bright-yellow colour. On microscopic section the cells are of large polygonal shape, arranged in palisade form. The protoplasm is clear, with large nucleus, contains highly refractile bodies, and is found to contain also both fat and glycogen.

Hypernephroma is disseminated both by direct extension and by metastatic deposit. If the growth reaches the renal vein, small emboli may attain the lungs and deposits may be found in the liver or in the bones, the latter being not infrequent.

2. *Primary carcinoma* of the kidney occurs as an infiltrating tumour in the substance of the organ. Formerly a large number of renal tumours were classified as carcinomata which now prove to be hypernephromata; carcinoma in this situation are uncommon. On microscopic section the growth may show an alveolar or a papillary arrangement of cells.

3. *Sarcoma* occurs both as the round- and the spindle-celled variety. It may attack the kidney in late adult life, but is more commonly a tumour of early years, occurring in quite

young children, in whom it may reach a large size. It arises in the capsule or in the tissues of the hilum, rapidly invading the renal tissue. On section it shows a greyish-white appearance with ill-defined periphery.

4. The *embryonic mixed tumours* show a great complexity of structure. They occur in the early years of life, arising from inclusions of embryonic tissue. Microscopically they may contain striped and unstriped muscle-fibres, cartilage, bone, and tubular or glandular epithelial elements, in a basis of elementary connective tissue. They have been classified as adeno-sarcoma (Birch-Hirschfeld) or as rhabdomyo-sarcoma, according to the structure that predominates. They are covered by a layer of compressed renal tissue, and may invade the renal vein.

Symptoms.—The prominent symptoms of new growth in the kidney are hæmaturia, pain, and the presence of a tumour.

Hæmaturia is usually the first symptom, and immediately attracts the patient's attention. The bleeding comes on suddenly without previous pain or exciting cause, lasts for one or several days, and then disappears for some days or weeks, to recur in the same form. In some cases hæmorrhage may follow a walk over rough ground, or a train journey. The bleeding may be profuse and accompanied by the passage of long, rounded worm-like clots with decolorized ends, indicating their formation in the ureter. In the intervals of hæmorrhage the urine is clear and free from albumin.

The intermittent, profuse hæmaturia is most common in hypernephroma. It is comparatively infrequent in the sarcomata of early childhood.

Pain is a common symptom and may be due to several causes. There is usually a constant dull aching in the loin, from tension in the renal capsule, with occasional exacerbations of more acute pain of a "tearing" nature, which is probably due to localized hæmorrhage into the growth. Constant aching pain in the loin may also be due to extension of the growth through the renal capsule, which may also cause pain in the distribution of the lower intercostal or lumbar peripheral nerves.

Pain may be due, further, to the passage of clots down the ureter, when it takes the form of acute colic, and will be followed by the passage of bloodclots per urethram.

Tumour in the loin is a frequent sign, especially in children, in whom it may be large

KIDNEY, TUMOURS OF

enough to form a visible prominence in the subcostal area.

Examination should be made with one hand in the loin below the twelfth rib and the other below the costal margin in the dorsal posture, and the patient instructed to breathe deeply; if no renal enlargement is detected, examination should be made in the knee-elbow position. The tumour may retain the shape of the kidney or, as is more frequent, present a localized, firm rounded mass from one aspect. The colon may be palpated in front of the swelling, or may give a band of resonance over an otherwise dull note to percussion over the main mass. The tumour moves with respiration and can be pushed up into the loin; if, however, the growth has extended into the perinephric tissues, movement is limited.

Occasionally a growth in the upper pole of the right kidney may displace the liver about its transverse axis, so that its anterior margin descends below the costal margin and renders palpation of the kidney difficult.

The remaining symptoms of the disease are due for the most part to pressure by the tumour or to metastases. Varicocele is occasionally present, and is especially significant if present on the right side or failing to disappear in the recumbent position. Œdema of the legs may be present from direct extension of the growth into the vena cava, or dyspnoea from pulmonary metastases. A skiagram of the renal area may show an increased opacity with ill-defined outline, while metastatic deposits in the lungs will also be shown by X-rays.

Diagnosis.—The occurrence of fairly profuse and sudden hæmaturia in an elderly patient, especially if accompanied by the passage of rounded, worm-like clots, is strong evidence of a renal growth. The only certain method of localizing the source of the hæmorrhage is by cystoscopic examination during a period of bleeding, when blood may be seen issuing from one ureteric orifice. The same examination will exclude any vesical lesion which might cause the hæmaturia.

The combination of renal tumour and hæmaturia is strong presumptive evidence of the renal source of the bleeding, but every case should be subjected to cystoscopic examination before any operation is contemplated. In more than one case in my experience the hæmaturia had occurred from a vesical lesion, which was so situated as to cause a partial obstruction to one ureteric orifice, from which the renal swelling (hydronephrosis) had resulted.

Other conditions causing a renal tumour and hæmaturia are always accompanied by other symptoms. Thus a renal calculus gives rise to pain and pyuria together with a well-defined radiographic shadow; tuberculosis is associated with pyuria and increased frequency of micturition in a younger patient, whilst in congenital cystic disease the swelling is bilateral and accompanied by the passage of large quantities of urine of low specific gravity.

When a renal tumour is present without hæmaturia, a localized swelling may be felt. The characteristics of a kidney swelling have been given, but care must be taken to differentiate it from a tumour of the liver, gall-bladder, spleen, or colon.

It is seldom possible to differentiate between the various forms of growth that may be present: sarcoma and embryonic tumours are more common in early years, hypernephroma and carcinoma in later life. Hypernephroma gives rise to sudden profuse hæmaturia in intermittent attacks, whereas in carcinoma the bleeding is more constant and less in amount. Pulmonary and osseous metastasis are more common in hypernephroma.

Treatment.—The only treatment holding any prospect of success is early and complete removal of the kidney. Before this is done, careful examination must be made that the growth has not extended beyond the limits of the kidney and that the other kidney is present and functionally active. The condition of the second kidney should be estimated by examination of the urine collected by ureteric catheterization and by the use of colour or phloridzin tests for renal function. The operation should be performed by the lumbar route, but if the tumour is large or any doubt exists as to peritoneal invasion, the peritoneum should be opened deliberately on the outer side of the colon and the organs thoroughly examined. Every attempt should be made to remove the perirenal adipose tissue with the kidney, and also the lymphatic tissue along the aorta and vena cava. This is most readily effected by the operation which has been described by Grégoire.

In cases in which operation is inadvisable, palliative measures may be necessary to control hæmorrhage or allay pain. Thus adrenalin, ergot, horse-serum, or calcium lactate may be given, with injections of morphia or scopolamine. The bowels must be regulated by suitable treatment.

LABOUR, MATERNAL INJURIES FOLLOWING

Prognosis.—The mortality from nephrectomy for renal growth has been greatly reduced in recent years by improved methods, and earlier operation should decrease the risk of recurrence. Recurrence may occur in the area of operation from escape of tissue-cells or by metastatic deposit in the lungs, liver, or bones. In the sarcomata of children recurrence is more rapid than in adults. In cases unoperated upon, death usually ensues within two years from the onset of symptoms.

R. H. JOCELYN SWAN.

KLEPTOMANIA.—A morbid desire or irresistible impulse to steal, in persons not tempted by needy circumstances, may be a manifestation of abnormal and degenerative mental states. It may be associated with other impulses or obsessions such as occur in psychasthenia. It is stated to be occasionally the result of an erotic fetishism.

GORDON HOLMES.

KRAUROSIS VULVÆ (see VULVA, DISEASES OF).

KYPHOSIS (see SPINAL CURVATURE).

LABIO-GLOSSO-LARYNGEAL PARALYSIS (see Bulbar Paralysis, under MUSCULAR ATROPHY, PROGRESSIVE).

LABOUR, FIBROIDS COMPLICATING (see UTERUS, NEW GROWTHS OF).

LABOUR, MATERNAL INJURIES FOLLOWING.—The common injuries which result from labour are: (1) rupture or laceration of the perineum, vagina, or cervix, (2) rupture of the uterus, (3) sloughing of the genital canal as a result of pressure, producing the various fistulae, and (4) rupture of a varicose vein in the vulva.

1. (a) **Rupture of the perineum.**—This injury occurs as a result of undue stretching of the vulval orifice at the moment of birth of the presenting part. It is most commonly produced at that moment during delivery when the foetal head is undergoing the extension movement, whereby a large diameter, namely the occipito-frontal, is suddenly thrown across the vaginal outlet. It is predisposed to by precipitate labour, when the soft parts have not had time to stretch, and may sometimes be the result of instrumental delivery. The shoulders very rarely tear the perineum when the head has not done so. In breech presentations the aftercoming head may tear the perineum, especially because rapid delivery of the head is necessary, and because the body of the child does not dilate the vaginal outlet sufficiently to allow the head to pass easily.

There are two varieties of ruptured perineum, namely, *incomplete* and *complete*, the latter meaning that the perineal body is torn right

through into the rectum, the sphincter ani being torn across. Rupture of the perineum is important for two reasons, first because it exposes a large raw surface to the possibility of infection; and second, because in severe cases it injures the pelvic floor, enlarging the aperture between the two levatores ani muscles and predisposing to prolapse of the uterus. For these reasons alone, it is important to try to prevent perineal lacerations, and, if they occur, to suture them at once so as to obtain primary union.

Prevention is often impossible, but an attempt should be made, in all doubtful cases, at least to limit the amount of laceration. The best way to do this is to keep the foetal head flexed as long as possible, by pressing with the thumb and second finger, on either side just behind the anus, upon the foetal forehead through the stretched pelvic floor tissues. At the same time the occiput may be pulled down, away from the pubes, with the other hand. In this way flexion is maintained and the birth of the head is delayed. The head can then be allowed to distend the vaginal outlet slowly, and is delivered with greater safety between contractions of the uterus than during a contraction. This is carried out by "expressing" the head, by pressure with the thumb and finger placed as mentioned above. The forehead, caught between the finger and thumb in this way, is pressed towards the pubes, and the occiput gradually emerges while the stretched perineum slips back.

If it is obvious that a laceration must occur, then it is good practice to make an incision in the middle line as far back as may be deemed

LABOUR, MATERNAL INJURIES FOLLOWING

necessary, so as to have a clean-cut instead of a bruised and lacerated surface to suture.

Such incisions rarely spread beyond their original extent, and give very good results. A central incision gives just as good results as the lateral incision which has been recommended in the operation of episiotomy.

Treatment.—All lacerations of the perineum, however small, should be sutured immediately, for if left alone they never heal by adhesion of their surfaces. The sutures should be so placed as to embrace the whole thickness of the perineal body, being entered on the skin, carried deeply through the tissues, and brought out through the vaginal mucosa, and continued in the reverse direction on the other side. The same principle should be carried out in complete tears, the posterior sutures including the rectal mucosa instead of the vaginal, and being also passed through the retracted ends of the sphincter ani. It is sometimes useful to suture the rectal mucosa separately with absorbable catgut.

(b) **Laceration of the vagina.**—Perineal laceration is often accompanied by laceration of the vagina, which may extend as high as the cervix, usually in the lateral angles of the vagina. More commonly on the left, these injuries cannot be seen without thorough opening up of the vagina with a speculum. They must be sutured at once with a continuous catgut stitch. If not sutured they give rise to troublesome bound-down cicatrices, as they always become septic.

(c) **Laceration of the cervix.**—This injury is usually the result of too early application of the forceps, but may occur spontaneously in precipitate labour, or be caused by the aftercoming head. It is often unrecognized, but should be thought of in every case of trickling post-partum hæmorrhage with a well-contracted uterus. In most cases it causes no symptoms, and is not discovered until months or years after. If it give rise to hæmorrhage or has been accidentally discovered, or deliberately looked for, laceration of the cervix should be sutured with absorbable catgut; a posterior speculum, a good light, and tenacula to pull down the halves of the cervix being required. The sutures are placed so as to embrace the whole thickness of the cervix, excluding the mucous membrane. These sutures will always stop hæmorrhage from a torn cervix, and it is unnecessary to attempt to look for and tie a bleeding vessel.

2. **Rupture of the uterus.**—The uterus is

usually ruptured as the result of obstructed labour, in cases either of malpresentation or of contracted pelvis. It is preceded, as a rule, by tonic contraction, and by dangerous thinning of the lower uterine segment, but this is not always the case. It may be caused by internal manipulations in an attempt to perform version, decapitation, or craniotomy in obstructed labour. This variety does not always cause immediate symptoms, but is usually recognized by the operator at once. Such cases, if treated at once, have a better prognosis than those which occur spontaneously. Uterine rupture nearly always starts in the lower segment, and thence may spread in any direction. It may be *complete*, when all the coats are involved, or *incomplete*, when the peritoneal cavity is not opened. The symptoms are a sudden feeling of something having "given way" inside, some external hæmorrhage, and collapse. The last is a variable symptom, as the pulse does not always rise rapidly. The fœtus may pass partly or completely through the rent into the peritoneum or folds of the broad ligament, the uterus contracting down and also expelling the placenta the same way. The diagnosis can be made by the sudden collapse, recession of the presenting part, hæmorrhage, and palpation of the fetal parts free in the abdominal cavity. If the rupture occurs during delivery *per vias naturales*, the symptoms are the same, but of course the fœtus is not in the abdomen.

Treatment.—One of three measures may be adopted—(a) Plugging the rent with gauze and providing drainage, (b) hysterectomy, or (c) suture of the rent. The first has given the best results in the past, the last has given the worst. Probably, in the future, with modern methods of shockless operating, hysterectomy will answer the best, provided that the patient is not in a desperate condition. Naturally, the treatment must depend upon the position of the child. If it has passed into the peritoneal cavity, the abdomen must be opened. Then the decision between the various alternatives must be influenced by the condition of the patient, the surroundings, and the skill of the operator.

3. **Fistulæ.**—See VAGINAL AND UTERINE FISTULÆ.

4. **Rupture of a varicose vein of the vulva.**—This sometimes occurs during labour. It may cause external bleeding if there is a tear, or it may cause a hæmatoma if no external tear occurs. Sometimes both take place,

LABOUR, PREMATURE, INDUCTION OF

only a small opening resulting. A hæmatoma may have widespread relationships, the hæmorrhage burrowing up into the pelvis in some instances. Resolution may follow, but these hæmatomata not infrequently become infected and suppurate. Incision with drainage is then the only treatment. In any case, if large, a hæmatoma should be incised, the clot turned out, and the cavity drained.

THOS. G. STEVENS.

LABOUR, PREMATURE, INDUCTION OF.

Induction of premature labour is the term applied to the artificial termination of pregnancy before the fortieth week and after the child has become viable.

The child is said to be viable after the twenty-eighth week, but its chances of survival are small if it is born before the thirty-fourth week; they are greatest after the thirty-sixth week. The longer the induction can be postponed the better for the child.

Induction is more risky for the child but less risky for the mother than Cæsarean section. The maternal mortality is low, and it is a procedure which can be carried out by the general practitioner. The disadvantage is that the child is prematurely delivered, and therefore its chances of survival are lessened.

It should be added that Cæsarean section in experienced hands as an alternative to induction has given excellent results.

Indications for induction. 1. Dangers to the mother.

Induction is indicated where Cæsarean section is contraindicated or refused: (1) In serious pathological conditions threatening the life of the mother, or causing permanent invalidism should pregnancy be prolonged, and when no improvement has taken place under appropriate treatment. Such conditions are—cardiac disease, when compensation has failed; pulmonary diseases, especially advanced tuberculosis; pyelitis; malignant disease of pelvic organs; toxæmias such as eclampsia, nephritis, accidental hæmorrhage, hyperemesis, and jaundice, when examinations of the urine and blood give indications for the termination of pregnancy; placenta prævia, to prevent recurrence of severe hæmorrhage; pernicious anæmia; leukæmia; diabetes, other than temporary glycosuria; chorea; goitre; insanity. (2) In pathological conditions of the ovum, and death of the fœtus in utero with septic absorption.

The results of induction in fevers such as typhoid and pneumonia are bad, as there is

increased risk to the mother even when premature labour occurs spontaneously. In considering the indications for induction, regard must be had both to the effect of disease upon pregnancy and to the effect of pregnancy upon a pre-existing disease.

2. *Dangers to the child.*—(1) *Moderate degrees of pelvic contraction*, especially in multipara whose labours have resulted in still-births. Induction is indicated where the true conjugate is not below $3\frac{1}{4}$ – $3\frac{1}{2}$ in., and in a generally contracted pelvis where it is not below $3\frac{1}{4}$ – $3\frac{1}{2}$ in. If it is less than 3 in., Cæsarean section or craniotomy must be performed. Labour is induced earlier in a generally-contracted pelvis, as the transverse diameter is lessened. Although rules are given for pelvic measurements, the time for induction depends upon the relative proportions between the pelvis and the fetal head, which should be systematically estimated weekly after the thirty-fourth week. As long as the head can be pushed through the brim of the pelvis, let pregnancy continue; but when the head is too large to enter the pelvis, induce.

(2) *Obstruction in the pelvis* due to tumours, rigidity of the cervix, or atresia of the vagina. But in such cases Cæsarean section is preferable to induction.

(3) *Habitual death of the fatus* from causes other than syphilis. Induce labour just before the date of expected death as ascertained from previous pregnancies.

Methods of induction. 1. Slow induction.—This gives the best results and imitates normal labour as nearly as possible. The cervix must admit one finger, dilatation having been effected by Hegar's dilators under anæsthesia or by the introduction of laminaria tents. If tents are selected, several small ones are inserted with forceps and, the vagina having been packed, are left in position for twelve hours.

All the methods employed for the induction of labour are uncertain if they are safe, but those most commonly employed are as follows:—

Rupture of the membranes is easy, but results frequently in a prolonged labour, and there is a risk of sepsis. It does not, as a rule, involve prolapse of the cord or malpresentations. Labour may be hastened by sweeping the forefinger round the internal os and partially separating the membranes in the lower uterine segment from the wall of the uterus, as occurs in normal labour.

LABOUR, PREMATURE, INDUCTION OF

Krause's method is that employed by most obstetricians, and gives good results. It consists in passing between the membranes and the uterine wall one or more gum-elastic bougies, sizes 10-12, which have been previously sterilized by boiling for ten minutes and placing in biniodide solution, 1:1,000. The patient having been anesthetized and placed in the lithotomy position, the vulva and vagina are sterilized and a posterior speculum is passed. The cervix is steadied with bullet forceps, and the plug of mucus removed by an iodide swab. One finger is passed into the cervical canal, and the membranes are gently separated from above the internal os. A bougie is passed up along the finger and introduced into the uterus between the membranes and uterine wall as far as possible. If resistance is felt, it is withdrawn and passed in another direction so as to avoid the placenta, which is usually situated on the posterior uterine wall. Care must be taken to avoid rupture of the membranes, or separation of the placenta, which may result in some bleeding. One large bougie may be used, or several smaller ones. If the ends project beyond the vulva they may be cut off.

The vagina is packed with gauze, which keeps the bougies in position, controls hæmorrhage, and has a reflex action upon the contractions of the uterus. If rubber catheters are used instead of bougies, the copper stylet must be withdrawn when introduction has been effected.

Labour may come on in 24-72 hours. The bougies may be left in position for 24 or 48 hours and then withdrawn and a new set introduced if labour has not occurred. When labour takes place they are expelled spontaneously.

If labour does not occur, it is better to introduce a Champetier de Ribes dilating bag. The medium-sized bag is sterilized by boiling while partially filled, its capacity and soundness having previously been ascertained. It is placed in biniodide or lysol solution, and introduced, folded, into the cervix by means of forceps, which push up its base beyond the internal os, between the membranes and the uterine wall.

The forceps are loosened, and some sterile water is introduced into the bag by means of a syringe; the forceps are withdrawn, and the bag is then filled with fluid and the tap turned, the end of the tubing being tucked up under the patient's abdominal binder.

If the labour has to be hurried, traction may

be employed by means of a 2-lb. weight attached to the end of the tubing and hung over a chair or towel-rail. The bag is left in position until expelled spontaneously into the vagina, when the fluid contents are run off.

If the membranes rupture, the bag prevents the loss of amniotic fluid.

The disadvantages of the bag are the difficulty of sterilizing it and of preserving the rubber. Further, it may displace the presenting part, and may conceal hæmorrhage if this occurs, with danger of subsequent rupture of the uterus.

If labour does not occur after the use of bougies and the dilating bag, podalic version may be performed and a leg drawn down. Traction on the foot outside the vulva will stimulate uterine contractions.

Plugging the vagina.—This method brings on labour and checks hæmorrhage in suitable cases of ante-partum hæmorrhage. It is said occasionally to cause rupture of the uterus in cases of toxæmia.

Intra-uterine injections stimulate the uterus and separate the membranes. Glycerin has a hygroscopic action on the liquor amnii, but has produced fatal toxic effects. Its use is not to be advised.

2. Rapid induction may be employed when the need is urgent, but these methods are risky in most cases, as they cause laceration of the cervix and lower uterine segment, and are followed by sepsis. They are useless in cases of pelvic contraction.

Accouchement forcé is the rapid and forcible dilation of the cervix, followed by the immediate expulsion of the child.

If rapid evacuation of the uterine contents is indicated, vaginal Cæsarean section before the twenty-fifth week, or abdominal Cæsarean section after the twenty-fifth week, is advised. Metal dilators, such as those of Bossi, are now seldom employed.

The safest of the rapid methods is manual dilatation when the cervix is softening and its canal obliterated. The patient is anesthetized, the hand is introduced into the vagina with one finger in the cervix, and the uterus is pushed down from above.

The thumb is then introduced into the cervix, followed by the second finger, until all the fingers lie within the cervix. They are slowly rolled upon one another and gradually expanded until the cervix is dilated. Some operators employ the index and second fingers of both hands and abduct them.

LABYRINTH, AFFECTIONS OF

Deep anæsthesia is necessary, and care must be taken to avoid deep lacerations.

It should be remembered that the more the dilatation is hurried, the greater the risk of laceration and sepsis.

A. LOUISE McILROY.

LABYRINTH, AFFECTIONS OF.—Perhaps the simplest method of discussing labyrinth diseases is to regard them from the point of view of symptoms. These fall into two groups: (1) those of acute labyrinth disease—the Menière syndrome or “labyrinth storm;” (2) those of chronic labyrinth disease.

In the former the most striking phenomenon is vertigo, from irritation or sudden paralysis of the vestibular system in the semicircular canals, whilst of the latter the chief feature is deafness, due to impairment of the auditory apparatus in the cochlea.

In acute labyrinth disease, however, involvement of the cochlea also occurs, for in practically all cases there is more or less tinnitus and deafness, while in chronic labyrinth disease the vestibular system also is attacked, as the slight occasional attacks of vertigo, coupled with the absence or enfeeblement of the vestibular reactions, plainly show.

Acute labyrinth disease is described under MENIÈRE'S DISEASE and need not be further considered here.

Chronic labyrinth disease.—The most prominent symptoms are tinnitus and deafness of the perceptive variety; but it should be noted that the vestibular system is often impaired also, even although vertigo may be but slight or altogether absent. (*See EAR, EXAMINATION OF.*)

A certain number of cases of chronic labyrinth disease begin suddenly and violently with an acute attack as described above. In the majority, however, the pathological process has so insidious an onset and progresses so gradually that little or no vestibular disturbance is experienced. The contrary is true of the hearing. No matter how gradual and imperceptible the onset of cochlear disease may be, sooner or later, if it advance, the patient will suffer from deafness.

Cochlear disease produces the severest types of deafness, for middle-ear disease alone never leads to absolute loss of hearing. In other words, severe deafness is always either purely perceptive in character or it is a mixture of obstructive and perceptive deafness. This latter type is quite common, for the most frequent cause of labyrinth disease (and of nerve-deafness) is middle-ear disease.

In addition to organic lesions of the nervous apparatus, a functional disability from disuse may supervene upon obstructive deafness; it is such cases, probably, which respond favourably to the many faith-healing and quack remedies that are on the market.

Common diseases of the labyrinth.—**Septic labyrinthitis** is of two varieties, the serous and the purulent.

Serous labyrinthitis is a not infrequent accompaniment of suppuration in the middle ear, especially when this is acute. It is supposed that the serous effusion is due to the infection in the middle ear, and that the labyrinth spaces themselves are not invaded by bacteria. The symptoms are of the acute type, and as pyrexia and pain may also be present the distinction between this and the graver purulent type is not easily made. Reliance is placed upon two circumstances: (1) the deafness, although it may be severe and perceptive in character, is not absolute; (2) although vertigo and spontaneous nystagmus may be, or may have been, present, the vestibular reactions are not abolished. In cases of general purulent labyrinthitis, on the other hand, the deafness is complete and the vestibular responses are lost.

Labyrinth fistula occurs with a circumscribed labyrinthitis when disease in the middle ear erodes the bone of the outer wall of the labyrinth without general infection of the labyrinth spaces. The chief symptom is a frequent, easily induced, and fleeting vertigo unaccompanied by the phenomena constituting the “labyrinth storm.” The vertigo may be artificially set up as follows: The nozzle of a Politzer bag is fitted closely into the external auditory meatus and the bag is firmly compressed so as to raise the air-pressure in the meatus. If the fistula be conveniently situated, the pressure is communicated through it to the labyrinth fluids, and sudden and often intense vertigo with violent nystagmus at once appears. When positive this sign, known as the “fistula symptom,” is of considerable value.

Acute purulent labyrinthitis is due to the invasion of the labyrinth spaces by pyogenic bacteria from the middle ear. Its onset is usually marked by the occurrence of the “labyrinth storm,” accompanied and followed by pyrexia (101° to 102° F.), and frequently by pain. After this initial warning, the disease often becomes latent, the pain and fever decline and disappear, and the patient and his physician may regard all danger as at an end. But

LABYRINTH, AFFECTIONS OF

during this latent stage there is absolute deafness in the affected ear together with an absence of the vestibular reactions, and sooner or later, if the disease be not relieved by operation, the latency terminates in acute septic meningitis and death.

Syphilis of the labyrinth usually appears as a late secondary phenomenon, but it may come on at any stage in the disease. It is characterized by the rapid onset of severe bilateral nerve-deafness with tinnitus and sometimes vertigo. After a gradual development the deafness becomes stationary for a period of weeks or months, and then a sudden change for the worse takes place which may end in total deafness. The diagnosis rests upon the presence of other signs of syphilis and upon the Wassermann reaction. Treatment is unsatisfactory. Pilocarpine hypodermically and blistering of the mastoid region have been lauded. The effect of the salvarsan compounds is not yet known, but it is feared that they may actually damage the hearing as a result of the Jarisch-Herxheimer reaction process (see under **SYPHILIS**).

Atrophic changes in the labyrinth are perhaps responsible for senile and other varieties of slow and invadescent deafness. They induce a quiet deafness without any vertiginous phenomena. Deafness of this kind is incurable, but temporary benefit may follow tonic doses of nux vomica.

Noise deafness (boilermaker's deafness, gun deafness) is due to the injurious effect of loud, painful, or continuous noise upon the cells of Corti's organ. It is incurable, but when the patient is removed from the malign influences it does not get worse. A measure of protection may be obtained by stopping the external meatus with plugs of plastic material (e.g. plasticine mixed up with cotton-wool fibres). Simple tampons of cotton-wool are more popular but are of little use.

Toxic deafness.—Salicylic acid and its salts, quinine, tobacco and alcohol, lead, mercury, arsenic, etc., may all lead to toxic deafness, that due to quinine being the commonest. For this reason, quinine is contraindicated in those whose hearing is defective.

DAN M'KENZIE.

LACTATION (see **PUERPERIUM, COURSE AND MANAGEMENT OF**).

LACTATION, INSANITY OF (see **PUERPERAL INSANITIES**).

LANDRY'S PARALYSIS

LACTOSURIA (see **URINE, EXAMINATION OF**).

LAGOPHTHALMOS (see **EYEIDS, AFFECTIONS OF**).

LANDRY'S PARALYSIS.—A form of acute ascending paralysis described by Landry in 1859.

Symptomatology.—The paralysis attacks previously healthy individuals without other prodromal symptoms than slight general malaise and paresthesia of the extremities. It begins as a flaccid paralysis of the legs, which are attacked in rapid succession. When the legs are completely paralysed the paralysis gradually involves the abdominal and spinal muscles, creeping higher day by day. A few days after the onset flaccid paralysis of the arms supervenes, next the muscles of speech and swallowing are implicated, and finally the muscles of respiration. The patient dies from respiratory failure after a period varying from four or five days to three weeks from the date of onset. In milder cases the disease becomes arrested before the respiratory muscles are reached, and the paralysis, after a varying period, begins to disappear in the reverse order to that of its onset. Sensation is never completely lost, but a certain degree of hypoaesthesia occurs *pari passu* with the paralysis. The deep and superficial reflexes disappear as the paralysis reaches their level. Pain is rarely complained of. The voluntary control of micturition and defecation is only occasionally lost.

Many varieties of acute ascending paralysis have been described, some with early bulbar symptoms or with involvement of the cranial nerves, but, inasmuch as the first description by Landry was a purely symptomatic one, nothing is to be gained by the inclusion of cases which do not conform to his description.

Pathology.—In the original cases described by Landry no positive pathological lesions were found. Of the large number of cases since described, some showed no lesion; some showed capillary hæmorrhages in the cord; others, disseminated inflammatory lesions in the medulla; and still others, degeneration of the anterior roots. It would appear that a large number of cases exhibiting Landry's syndrome are due to an ascending acute anterior poliomyelitis, a smaller number to an ascending neuritis, and most of the others to different forms of ascending meningeal infections.

Treatment.—Most of the cases run a fatal course, no matter what form of treatment is adopted. It is essential to decide, if possible, to what the paralysis is due, and to treat the primary infection if it can be discovered. Repeated lumbar puncture is useful in some cases, particularly in those of meningeal origin, and large doses of urotropine should be administered. Oxygen, and perhaps artificial respiration, may be required if respiratory embarrassment sets in.

F. L. GOLLA.

LARDACEOUS DISEASE (see AMYLOID DISEASE).

LARGE WHITE KIDNEY (see NEPHRITIS).

LARYNGEAL PARALYSIS (see VOCAL CORDS, PARALYSIS OF; and Bulbar Paralysis, under MUSCULAR ATROPHY, PROGRESSIVE).

LARYNGEAL STRIDOR (see STRIDOR).

LARYNGISMUS STRIDULUS. A symptom due to spasmodic closure of the glottis, producing a period of apnoea followed by a crowing inspiration.

Etiology.—Laryngismus is practically confined to rachitic infants. It is found particularly in those showing marked hyperexcitability of the nervous system (spasmophilia). Thus it is almost always associated with facial irritability (Chvostek's sign), often with tetany or convulsions, and occasionally with head-nodding. Probably intestinal derangement predisposes to laryngismus in rickety subjects. In older children it is occasionally seen in association with tetany (q.v.), in which connexion it is interesting to note that bulbar symptoms are common in the tetany of adults.

Exciting causes of an attack are outbursts of passion or crying, exposure to cold, or the regaining of consciousness after sleep.

Symptoms. There is a sudden cessation of breathing, the child possibly turning a little pale, or even dusky. The apnoeic period usually lasts four or five seconds, at the end of which time the spasm relaxes and there is a deep inspiration accompanied by a crowing sound as in whooping-cough. In the usual slight case the crowing is the most noticeable feature, no anxiety being caused by the apnoea. More severe symptoms, deep cyanosis, unconsciousness, and convulsions are rare, but even fatal asphyxia is not unknown. The attacks may be repeated very frequently.

Diagnosis.—Parents often regard laryngismus as caused by the child "holding its breath" in a fit of passion, but once an attack has been seen or heard the diagnosis is easily made. The history should serve to differentiate laryngismus from *congenital laryngeal stridor*: while the absence of hoarseness, cough, fever, and true dyspnoea excludes such conditions as *catarrhal laryngitis* with super-added laryngeal spasm (*laryngitis stridulosa*) and *laryngeal diphtheria*. It should be remembered that laryngismus is a purely spasmodic condition in which there is no underlying laryngitis, and that it is associated with apnoea rather than dyspnoea.

Prognosis.—As a rule the attack is very mild, giving rise to little anxiety and quickly amenable to treatment. Serious cases are uncommon and fatalities very rare.

Treatment.—The infant must be kept very quiet. It should rapidly be put under the influence of bromide, 3 gr. of sodium bromide being given three times a day for a child of 6-12 months old. In severe cases this may be reinforced by chloral hydrate, given in the same doses as the bromide, or by tinct. opii in doses of $\frac{1}{4}$ min. for each three months of the child's age. A dose of castor oil may also be given at the outset of treatment.

In alarming attacks the child may be immersed in a hot bath, or its face sponged with hot water. If cyanosis is severe an effort to relax the spasm may be made by passing the finger into the back of the mouth as in attempting to produce vomiting. But usually the attack itself requires no immediate treatment.

REGINALD MILLER.

LARYNGITIS.—An inflammatory condition of the mucous membrane of the larynx. The following forms may be enumerated:—

1. Acute.

- (a) Catarrhal. Spasmodic.
- (b) Septic. Oedematous.
- Membranous.

2. Chronic.

- (a) Simple.
- (b) Sicca.
- (c) Atrophic.
- (d) Hyperplastic.
- (e) Pachydermatous.
- (f) Subglottic.
- (g) Granular.

For Tuberculous Laryngitis, see LARYNX, TUBERCULOSIS OF. For Syphilitic Laryngitis, see LARYNX, SYPHILIS OF.

LARYNGITIS

Etiology.—The causes may be divided most conveniently into predisposing and exciting.

Predisposing.

1. Mouth-breathing, as in nasal obstruction.
2. Sepsis in the upper respiratory area.
3. Bad hygienic conditions, such as obtain in ill-ventilated rooms and dusty occupations.
4. Abuse of alcohol and tobacco.
5. Rheumatism and gout.
6. Improper voice-production.

Exciting.

1. Infection by a definite micro-organism — streptococcus, pneumococcus, etc.
2. Traumatism.
 - (a) Passage of foreign bodies or instruments.
 - (b) Scalds.
 - (c) Irritating fumes.
3. Specific fevers.

ACUTE CATARRHAL LARYNGITIS

Symptoms.—There is usually a slight rise of temperature, and invariably a hoarseness (slight, or leading to complete loss of voice), with an irritating dry cough, and a sense of discomfort in the throat. As the case progresses the inflammation spreads down the trachea, when the secretion becomes profuse, and the cough looser, with abundant expectoration. On examination the mucous membrane is seen to be congested and both cords are symmetrically red and injected. If the condition persist for some time, small patches of erosion make their appearance, and the surfaces of the cords become rounded instead of being flat, the maximum swelling being about the middle of each cord and resulting in their imperfect approximation. In *children* the accompanying dyspnoea and constitutional disturbances are much more pronounced. A special type in children is *spasmodic laryngitis*, in which there is a marked tendency to spasm of the glottis. The spasms occur chiefly at night, and are accompanied by severe inspiratory stridor, with symptoms of asphyxiation. The severity of the spasms increases more and more until the child becomes almost collapsed, when they suddenly cease and the exhausted patient falls into a deep sleep.

Diagnosis is based on the history of the sudden onset, accompanied usually by symptoms of a cold in the head, the slight rise of temperature, the hoarseness, and the bilateral inflammatory affection of the cords.

Prognosis.—The condition generally clears

up in about a week under treatment, but if any strain be put upon the voice during the attack it may pass into the chronic form. In children it is of more serious import owing to the smallness of the larynx and the looser nature of the mucous membrane, which more readily lends itself to oedema.

ACUTE SEPTIC LARYNGITIS

In this form the **symptoms** are more pronounced and are ushered in by rigor and a high temperature. Free perspiration takes place, and hoarseness, pain, cough, dysphagia, and dyspnoea are usually present. On examination, in the early stages the mucous membrane is seen to be dark-red and swollen, particularly in the region of the epiglottis and aryepiglottic folds. Later, ulceration with or without hæmorrhage, and abscess formation with necrosis may take place, septic pneumonia being a likely danger. The **prognosis** is serious—death may take place early from cardiac failure or from oedema of the larynx, or later from pneumonia.

An *acute membranous laryngitis* may occur as a variety of this disease, a greyish-white membrane, firmly adherent to the subjacent mucosa of the larynx, being formed. It is non-diphtheritic, but of grave prognosis. *Acute oedematous laryngitis* is another variety in which marked dyspnoea, due to the oedema, is the special feature.

Treatment.—In the *acute catarrhal* form confine the patient to bed, keep the temperature of the room at 65° F., and forbid the use of the voice. Begin with a smart purge, calomel followed by a saline being the best. I have had excellent results with hexamethylene-tetramine, 10 gr. every four hours in a glassful of water.

Locally, inhalations of tinct. benzoini co., 1 dr. to a pint of water at 140° F., are useful, as also is the following:—

℞ Menthol gr. x.
Chloretone gr. iv.
Paroline ʒi.

This is used in a fine atomizer with a throat-piece. If there be an accompanying nasal catarrh it can also be sprayed into the nasal passages.

For much pain and an irritating cough a linctus containing heroin $\frac{1}{2}$ – $\frac{1}{4}$ gr. is beneficial.

For the *spasmodic laryngitis* of children, confine the patient to one room and commence with a purge. Hot compresses over the larynx

LARYNGITIS

are soothing, and a steam kettle, to the water of which some tinct. benzoini co. has been added, may be used with advantage.

Bromide should be given thrice daily. If there be much cough with free mucus, 60 min. of vinum ipecacuanhæ as an emetic is often very helpful. During a spasm, place the child immediately in a hot mustard bath, and give 1-2 min. of nitrite of amyl as an inhalation. Intubation and tracheotomy are rarely needed.

In **acute septic laryngitis** the treatment indicated above for the acute catarrhal type should be initiated. Here, however, quinine is a good internal remedy. The maintenance of the strength by a nutritive diet is very important. An early dose of 10 c.c. of antistreptococcic serum should be administered as a routine. In the early stages a Leiter's coil or cold compress over the larynx is useful.

Any symptoms of cardiac failure must be combated by digitalin and strychnine hypodermically. For oedema, a spray of 1-in-2,000 adrenalin with 2-per-cent. cocaine may be tried, and, if this fail, scarification should be performed.

In many of these cases life has only been saved by tracheotomy, and the necessary instruments should be kept at hand. Whenodynphagia is present the insufflation of orthoform or anæsthesin powder gives great relief.

CHRONIC LARYNGITIS

Etiology.—Chronic laryngitis frequently follows an acute attack, but it may occur primarily, three chief factors contributing—mouth-breathing, faulty voice-production, and suppuration in the upper respiratory passages.

Symptomatology.—The voice is husky or hoarse, and a dry cough, the result of laryngeal irritation, is present. On examination the vocal cords are usually found to be reddened and injected; blood-vessels may be seen on their surface, and sticky strings of mucus lying on the cords, or stretching across the glottis, may be present. Other cases show a considerable hyperplasia of the mucous membrane, the ventricular bands being so thickened that they may hide the true vocal cords. These are examples of the *hyperplastic* variety.

When the thickenings are limited to the vocal processes and the interarytenoid commissure the condition is known as *pachy-*

dermia laryngis. Again, the whole mucous membrane may present a dry, red, glazed appearance, to which the name of *laryngitis sicca* is applied; and when in addition there is some ulceration, with the presence of dark-grey or green crusts on the vocal cords, and especially in the interarytenoid commissure, the condition is called *atrophic laryngitis*. This is frequently associated with a similar condition of the nose.

In *granular laryngitis* the mucous glands in the arytenoids and aryepiglottic folds enlarge and become prominent, appearing as small projecting granules.

In the *subglottic* variety swellings can be seen beneath the vocal cords encroaching on the lumen of the trachea, and dyspnoea is one of the chief symptoms.

Diagnosis.—The points to which particular attention must be directed are the chronicity of the condition, the absence of general constitutional symptoms, and the bilateral affection of the vocal cords. *Pachydermia laryngis* may closely resemble *tuberculous laryngitis*, but in the latter the thickenings in the interarytenoid commissure are more gelatinous, softer, and more irregular; moreover in *pachydermia* the protuberances of one side fit into depressions on the other on adduction of the cords. In *syphilitic laryngitis* the edges of the erosions are more sharply cut and hyperæmic, and have a punched-out appearance.

In doubtful cases, examination of the chest and sputum, and Wassermann's reaction, will prove of considerable value. For further diagnosis, see LARYNX, NEW GROWTHS OF, also LARYNX, SYPHILIS OF, and LARYNX, TUBERCULOSIS OF.

Treatment.—First remove such predisposing causes as nasal obstruction, bad conditions of hygiene, over-use of tobacco and alcohol, and faulty voice-production. Then vocal rest must be enjoined and the patient instructed to speak in a whisper or lower vocal register until all symptoms have disappeared.

Internally, a combination of iodide of potassium with nux vomica and arsenic may prove of considerable value.

Locally, menthol administered by an atomizer is useful, or a more stimulating inhalation may be tried, such as oil of Scotch pine, 1 dr. to a pint of water at 140° F. Another satisfactory remedy is a 10-per-cent. solution of protargol given as a fine spray. *Cataphoresis*

and vibration over the larynx have proved of great benefit in many cases, as also a poultice of equal parts of mustard and linseed over the thyroid cartilage.

In laryngitis sicca and in the atrophic variety, iodides and expectorants internally and oily sprays locally prove most efficacious.

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LARYNGOSCOPY.—For the adequate examination of the larynx the following aids are necessary: A good light—the best is undoubtedly the Ediswan Pointolite, but any good flame with a suitable condensing lens will answer. A forehead mirror, which should have a short focal length of about 10 in. and a large round aperture; it can be worn either with a forehead band or set on a spectacle frame. Laryngoscope mirrors, which are set on their own handles at an angle of 120°, should be of three sizes, of diameters of $\frac{1}{2}$ in., 1 in., and $1\frac{1}{2}$ in. It is always advisable to use the largest size possible without causing irritation.

The patient being seated opposite to the examiner, with the light over his left shoulder and on a level with the examiner's forehead, he should be directed to open his mouth and put out his tongue. This should be held out gently but firmly with a tongue cloth in the left hand, care being taken not to press it down too much on the lower incisor teeth. The beam of light should then be directed on to the palate and fauces, and, having been warmed over a spirit lamp, and first tested on the examiner's own hand to see that it is not too hot, the laryngoscopic mirror, held in the right hand like a pen, should be passed into the mouth until the back of the mirror rests on the soft palate at the base of the uvula. The soft palate and the uvula should then be carried gently upwards and backwards until almost touching the posterior wall of the pharynx, and this done, the patient is directed to say "e—e," so as to elevate the larynx and bring it into view. A systematic examination can now be carried out, commencing with the interarytenoid commissure, and comprising the arytenoids, the aryepiglottic folds, the epiglottis, the ventricular bands, and the vocal cords, both as regards their appearance and their movements. Occasionally difficulty arises from the presence of an overhanging epiglottis; in this event the patient must be made to hold out his tongue with his right hand, and, after cocaineization, the examiner

with his left hand passes in a bent probe over the epiglottis and hooks this back, examining as before.

The usual mistake made in laryngoscopy is that of placing the mirror too far forwards on the soft palate, and pressing back the whole structure on to the posterior wall of the pharynx, producing immediate hawking.

In the case of a patient with an extremely irritable pharynx, it is advisable first to spray the palate and pharynx with a 5-per-cent. solution of cocaine.

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LARYNX, CHONDRITIS AND PERICHONDRITIS OF.—A condition of inflammation of the laryngeal cartilages and perichondrium.

Etiology.—There are two main types, acute and chronic. The *acute* form may be caused by (a) certain infectious diseases such as typhoid, typhus, diphtheria, variola, pyæmia, and scarlatina; (b) trauma, resulting from blows or wounds, or arising internally as from foreign bodies within the larynx. Traumatic chondritis has been known to follow the passage of œsophageal bougies, the œsophagoscope and bronchoscope. It may also be produced in old people if there is considerable friction between the ossified cartilages and the vertebral column; this chiefly occurs when they are confined to bed, in the dorsal decubitus. The *chronic* form is seen chiefly in tuberculosis, syphilis, and malignant disease, but may also result from an infection of low virulence, secondary to some slight traumatic lesion.

Pathology.—In the early stages of the acute type, considerable œdema and redness of the affected part are seen. An exudation of fluid takes place between the cartilage and the membrane, separating the latter from the former. This effusion becomes rapidly purulent, with the formation of an abscess, which may rupture either externally or internally according to the site of formation, and be followed by fistulæ, which may exude pus for a considerable period. After the separation of the membrane, the cartilage undergoes necrosis and subsequently separates, a marked deformity being produced by the falling in of the soft parts, and the subsequent contraction of cicatrices. Loss of voice may result, and stenosis, causing marked difficulty in respiration.

Symptomatology.—In the acute form the onset is usually accompanied by malaise, pain,

rigors, and a rise of temperature to 101°-102° F.; in the less acute, the early symptoms are pain and great tenderness over the whole laryngeal area. According to the position affected, other symptoms such as cough, hoarseness, aphonia, dyspnoea, and dysphagia may arise. When abscess-formation takes place the acute symptoms abate after its rupture, and bare cartilage may be felt by probing. The dead cartilage is exfoliated, and either coughed up or swallowed. Occasionally the sequestrum becomes impacted in the larynx, leading to severe or even fatal dyspnoea.

Diagnosis.—The acute septic form must be diagnosed from acute oedema of the larynx. Before abscess-formation this is difficult, but the constant pain and tenderness of the former with its more localized swelling are points of importance. The associated symptoms of the primary affection help to distinguish the more chronic types met with in syphilis, tubercle, and malignant disease. In the syphilitic type there are usually absence of pain, a positive Wassermann reaction, and improvement with iodides. Between the tuberculous and malignant forms the diagnosis is sometimes far from easy, but in the tuberculous there are usually definite pulmonary symptoms, and an examination of the sputum may show tubercle bacilli, whilst a small portion of cartilage can be removed for microscopical examination, to settle any question of malignancy.

Prognosis.—In acute cases death may occur from dyspnoea, septicæmia, pyæmia, or septic pneumonia, whilst, at the best, extensive damage will result to the larynx, with alteration in the voice and difficulty in breathing. In the tuberculous and malignant types the prognosis is grave.

Treatment.—In the acute cases, put the patient to bed and administer a strong purge—calomel (5 gr.) at night, with a saline in the morning. A Leiter's coil should be applied locally and ice constantly sucked. Steam inhalations at a temperature of 140° F., with either tinct. benzoini co. or menthol added, should be employed.

If the disease is external to the thyroid cartilage and fluctuation is present, the swollen area should immediately be incised under a local anæsthetic such as ethyl chloride, and drained. When it is internal and considerable swelling is present, the swollen area should be painted with a 10-per-cent. solution of cocaine, and incised. If an abscess has formed, this should be opened under local anæsthesia, but

on no account must a general anæsthetic be given. When the dyspnoea is very pronounced it may be necessary to perform tracheotomy, the instruments for which should always be kept at hand.

When the condition has arisen from the impaction of a foreign body, this must be removed forthwith. It is often necessary to perform a preliminary tracheotomy and remove the foreign body under a general anæsthetic. Sequestra should be removed at the earliest opportunity, and if it is found impossible to do this through the mouth, thyrotomy is indicated.

In the chronic types local treatment must be directed towards the alleviation of pain, and general treatment given to the primary disease.

In all cases the strength of the patient must be maintained as far as possible by a generous diet of semi-solids and fluids by the mouth, and by rectal alimentation. In the chronic septic types quinine is of considerable value.

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LARYNX, ENDOSCOPIC EXAMINATION OF (see RESPIRATORY PASSAGES, ENDOSCOPIC EXAMINATION OF).

LARYNX, FOREIGN BODIES IN (see FOREIGN BODIES IN THE AIR-PASSAGES).

LARYNX, INFLAMMATION OF (see LARYNGITIS; LARYNX, CHONDRITIS AND PERICHONDRITIS OF).

LARYNX, NEW GROWTHS OF.—The tumours met with in the larynx are conveniently divided into (1) benign and (2) malignant.

1. BENIGN OR INNOCENT TUMOURS

These will be considered in one group, as they cause much the same symptoms and require very similar treatment. They are more frequently found in the larynx than malignant tumours, but even so they are comparatively rare. It took Morell Mackenzie eight years to collect his first hundred cases.

Varieties.—The following is a list of the more usual innocent growths met with in the larynx, arranged approximately in order of frequency: Papilloma, fibroma, cysts, lipoma, angioma, adenoma, myxoma, lymphoma, enchondroma, thyroid-gland tumours, amyloid tumours. All, except the first two, are very rarely met with.

Papilloma is the most common. It occurs

LARYNX, NEW GROWTHS OF

at all ages, but is most frequent in infancy and childhood. It may grow from any part of the larynx, except the interarytenoid region. Papillomata may be single or multiple (Figs. 44 and 45). When solitary the growth may be white, pinkish, or even bright red. It may be broad-based or more or less pedunculated. It



Fig. 44.—Papilloma of the larynx in the adult.

does not ulcerate, nor does it invade the tissue from which it springs. *Fibroma*, which comes second in order of frequency, is found at any age, but is perhaps most common from 30 to 50. It may occur anywhere, but, whilst



Fig. 45.—Papillomata of the larynx. (St. Thomas's Hosp. Mus., No. 1791b.)

rare on the ventricular bands or epiglottis, commonly affects the vocal cords as a single growth, sessile or pedunculated, with a smooth or rough surface and greyish-white, pink, or even dark-red colour. *Cysts* are most common on the epiglottis (Fig. 46) and rare on the cords, while they may be found on the ventricular bands and aryepiglottic folds. The remaining

benign tumours are so rare that a description of them should be sought in special textbooks.

Etiology.—The cause of benign growths in the larynx, as in other situations, is uncertain. They are most frequently met with between the ages of 20 and 50, and seem to affect males more frequently than females. Chronic catarrh and misuse of the voice are often invoked as etiological factors, but without justification. A papilloma may develop before speech does, and may even be congenital.

Symptomatology.—The symptoms vary greatly in different cases and at different ages. When the growth occurs on the epiglottis or the pharyngeal surface of the aryepiglottic fold, it may cause no symptoms at all and be discovered by accident. When implanted on a

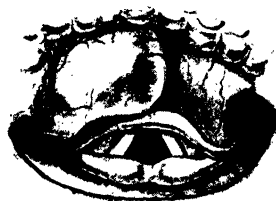


Fig. 46.—Cyst of the epiglottis.

vocal cord, a growth produces discomfort, vocal fatigue, and hoarseness, all of which are proportionate to its size and situation. When a growth is large, as may occur with a lipoma, or multiple, as with papillomata, the symptoms of dyspnoea, stridor, and spasmodic laryngitis may be added. Stridor is always an indication for operation. Cough is uncommon, and dysphagia very rare. There is neither pain nor general disturbance of health.

Diagnosis is based on the sex and age of the patient, the history of the case, the characteristics of the tumour, and the inspection of the larynx. The latter can nowadays be carried out in even the smallest patient, thanks to Killian's invention of direct laryngoscopy. In a few cases complete diagnosis may only be arrived at by the removal and microscopical examination of the growth. The following table indicates the points of difference between a simple and a cancerous growth in the larynx:—

LARYNX, NEW GROWTHS OF

BENIGN

Rare after 50.

Occurs on the anterior two-thirds of the vocal cords in the majority of cases.

Grows away from the tissues.

Base of growth, or cord it springs from, only inflamed during laryngitis.

May impair action of cord mechanically.

No ulceration.

MALIGNANT

Rare under 40; and generally occurs over 50.

A solitary growth on the aryepiglottic folds, the epiglottis, or on the cords is very suspicious in a patient over 45.

Invades the tissues.

Inflamed base.

Any impairment due to infiltration is strongly suspicious.

Tendency to ulceration.

Prognosis.—Many innocent growths remain stationary for years, and some patients who are not professional voice-users are content to put up with a certain amount of discomfort and chronic hoarseness. Papillomata in children may spread and increase so greatly as to cause voicelessness and threaten suffocation, whilst, on the other hand, they sometimes tend to atrophy and spontaneous disappearance. In adults complete removal of a benign growth is not followed by recurrence, and the prognosis in relation to voice is good.

Treatment.—Most cases call for operation, and, a few large extrinsic ones excepted, all laryngeal growths can nowadays be removed through the mouth. This can be done in adults under cocaine, and either by the indirect or the direct method of laryngoscopy.

Papillomata in children require separate consideration (*see above*). If there be no decided laryngeal stenosis, it is well to defer operation and try the effect of the prolonged administration of arsenic, iodide of potassium, or calcined magnesia, while any adenoids present are attended to. If there be stridor, suffocative attacks, or threatened loss of voice, the growths should be removed, under cocaine or chloroform, by Killian's direct method or by his suspension laryngoscopy. Should these methods not be available, a low tracheotomy must be performed. The tube may need to be worn for two or more years, when the papillomata tend to disappear spontaneously, but, in the meantime, further efforts at removal should be made by direct laryngoscopy.

Laryngo-fissure for these benign growths is quite uncalled for.

2. MALIGNANT TUMOURS OF THE LARYNX

Malignant disease of the larynx is not common. Carcinoma, and the still rarer sarcoma, will be considered together, as they are clinically indistinguishable.

Etiology.—There is no justification for blaming heredity, catarrh, over-use of the voice, tobacco or alcoholic excess as predisposing factors, although there is more reason for suspecting syphilis in this respect. Men are much more liable to laryngeal cancer than women. With rare exceptions it is not met with below the age of 40; it is most frequent between 50 and 60, and may occur in old age.

Clinical classification.—It is useful to classify cancer of the larynx into (a) *intrinsic*, i.e. originating within the cavity of the larynx, and (b) *extrinsic*, i.e. starting on the upper margins or outer walls of the larynx—the epiglottis, arytenoids, aryepiglottic folds, the pyriform sinuses, and the pharyngeal surface of the larynx (postericoid carcinoma). In addition, we may consider a group as (c) *mixed*, i.e. a combination of extrinsic and intrinsic, and covering the large number of cases which, unfortunately, only present themselves in an advanced stage.

Intrinsic cancer, which might more definitely be called "vocal-cord carcinoma," is very slow-growing in its early stages; it does not affect the lymphatic glands until late, and secondary growths are almost unknown. Fortunately, it declares itself early by slight but constant hoarseness. It is the more common variety, and prompt operation gives better and more lasting results than can be claimed for cancer in any other part of the body. With extrinsic cancer the conditions are very different. In the early stages the symptoms are uncertain or even absent; the voice may be unaltered and swallowing unaffected; glands are soon invaded; the progress of the disease is rapid, and it is seldom checked or cured by operation.

Symptomatology.—The symptoms given in older textbooks, dyspnoea, dysphagia, hæmorrhage, salivation, pain in the ear, fætor, wasting, cachexia, only indicate a late, and generally hopeless, stage of the disease. The early indications of a growth vary according to its situation: if on a cord, the chief symptom is obstinate hoarseness; if elsewhere, only local discomfort may be complained of. When per-

sistent, either of these symptoms calls for an expert examination of the throat.

Early appearances are very variable and often difficult to recognize. Nothing may be visible at first beyond a local congestion with some thickening. Later on we may encounter a definite tumour, an irregular infiltration, or a fungating growth. The last is generally irregular, greyish, dusky red, or dirty white, and ulcerating. The growth tends to infiltrate, so that, when it is situated on a vocal cord, the movement of the latter may be interfered with. The glands in the extrinsic variety are so early involved that a tumour in the neck may be the first thing a patient complains of; but it is very important to remember that in cancer of a vocal cord a diagnosis must be arrived at long before there is any glandular enlargement.

Diagnosis.—Cancer of the larynx may be confounded with chronic laryngitis, benign tumour, pachydermia, bloodclot, laryngeal palsy, perichondritis, syphilis, and tuberculosis. The diagnosis between an innocent and a malignant growth is shown in the table on p. 171. A one-sided congestion or infiltration is always suggestive of cancer, tubercle, or syphilis. A growth in a subject over 40 is more often malignant than innocent, and this probability is increased if it infiltrates a vocal cord so as to impede free abductor movement, or if it shows a surface like a short-cropped, white meadow. Tuberculosis favours the posterior half of the larynx, its victims are generally the young, and a careful search will reveal in most cases the pulmonary infection of which the laryngeal condition is but a secondary manifestation. Syphilis may invade any part of the larynx, with perhaps a slight preference for the epiglottis. The history and appearances, together with laboratory tests, will generally distinguish it; but it is important to remember that cancer grows well on a syphilitic soil, and that the two diseases may be present at the same time.

In some cases the removal of a good portion of the growth for microscopical examination may be possible.

Prognosis.—If untreated, cancer of the larynx inevitably ends in death by asphyxia, dysphagia, hæmorrhage, sepsis, or intercurrent complications. The average duration is one to three years. With operation the progress of extrinsic cancer may be delayed, and a few lasting cures have been obtained; but, once the lymphatics are invaded, recurrence is

almost certain. With intrinsic cancer of the vocal cords the prognosis is completely changed by early operation. Butlin, Semon, Schmiegelow, Chevalier Jackson, and StClair Thomson have all published records which show a lasting cure in 70 to 80 per cent. of cases, and this result is secured with the preservation of a natural voice, a restoration to working health, and with a death-rate of only 1-4 per cent. from operation.

Treatment.—The only radical treatment is surgical, and this, when possible, should aim at excision of the tumour with a surrounding area of healthy tissue, and the extirpation of all glands if involved. This may have to be carried out by (a) intralaryngeal removal, (2) thyrotomy (laryngo-fissure), (3) partial laryngectomy, (4) subhyoid pharyngotomy, or (5) total laryngectomy.

The first method has occasionally succeeded, but is no longer justifiable in face of the safe and brilliant results secured by laryngo-fissure. This latter operation, unfortunately, is only suitable for cancer of the vocal cord when diagnosed in good time. For a consideration of this and other methods the reader may be referred to the writer's textbook ("Diseases of the Nose and Throat").

Palliative treatment is directed to keeping the patient as free as possible from pain and discomfort, disinfecting the mouth and throat, checking hæmorrhage, and performing tracheotomy or gastrostomy if required. Neither radium nor diathermy gives much help in this particular region. Intramuscular injections of arsenic, or of the colloidal preparations of selenium or copper, sometimes appear to slow the progress of the disease and keep the parts in a cleaner state.

STCLAIR THOMSON.

LARYNX, OEDEMA OF.—A swelling produced by infiltration of the submucous connective tissue of either the whole or part of the larynx.

Etiology.—Injury, thermal, chemical, or mechanical, is a cause. The condition may accompany infectious fevers, or be secondary to acute septic infections in surrounding structures, such as pharyngitis, carcinoma of the œsophagus, tonsillitis, peritonsillar abscess, and Ludwig's angina. It may also occur in malignant, syphilitic, and tuberculous disease of the larynx, and in certain general diseases, such as Bright's disease, diabetes, and cardiac disease. It is occasionally met with after administration of potassium iodide.

LARYNX, ŒDEMA OF

Pathology.—The œdematous area appears inflamed and swollen, with translucent edges. The whole larynx may be affected, but usually the swelling is limited to one area such as the arytenoids, the aryteno-epiglottic folds, the epiglottis, the ventricular bands, or the subglottic region. When the arytenoids and aryteno-epiglottic folds are involved the lumen becomes encroached upon, the swellings appearing as two sausage-shaped masses, one on each side of the larynx.

Symptoms.—The onset is sudden, with pain and discomfort in the throat, and fever which may be ushered in by a rigor. The temperature rises to 100°–103° F. The voice alters early, becoming hoarse, and later being lost. Dysphagia and dyspnoea with stridor may rapidly supervene in acute septic cases. In more chronic examples, such as occur in diabetes, the onset is more gradual, the chief symptoms being some dysphagia with hoarseness and discomfort in the throat.

Diagnosis.—The sudden onset, the localized symptoms, and the œdematous swellings usually make this clear. In the chronic variety the history and the laryngoscopic appearance of pale translucent swellings indicate the condition.

Prognosis.—In severe cases this is always grave; death may occur from suffocation, cardiac failure, or pneumonia.

Treatment. General.—Put the patient to bed and open the bowels thoroughly; if there is much dysphagia, feed rectally. Two most valuable drugs are quinine sulphate 5 gr. t.i.d.s. and tinct. ferri perchlor. 30–40 min. t.d.s. Stimulants, strychnine, and digitalin must be used to combat cardiac failure. In bad septic cases antistreptococcal serum should be employed.

Local.—In the first instance apply Leiter's coils or ice-bags to the neck. A laryngeal spray of 2-per-cent. solution of cocaine with 1-in-2,000 adrenalin gives relief. If the œdema increases, infiltrated parts should be scarified under cocaine anaesthesia. The onset of marked dyspnoea indicates the need for an early low tracheotomy. (Edema due to potassium iodide is best combated by the administration of large doses of sodium bicarbonate. If there is any lung complication, oxygen should be given freely.

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LARYNX, PARALYSIS OF VOCAL CORDS OF (see VOCAL CORDS, PARALYSIS OF; and Bulbar Paralysis, under MUSCULAR ATROPHY, PROGRESSIVE).

LARYNX, SYPHILIS OF

LARYNX, SPASM OF (see LARYNGISMUS STRIDULUS).

LARYNX, SYPHILIS OF.—*Inherited* syphilis of the larynx is rarely seen. In early life it occurs as a perichondritis, later in life as various forms of hyperplasia.

ACQUIRED LARYNGEAL SYPHILIS

A **primary sore** is extremely rare. Even **secondary** manifestations of acquired syphilis seldom come under observation.

Pathology.—The lesions of secondary syphilis which may be met with are an erythema or catarrh, the whole larynx being congested and of a dusky patchy redness, and, rarely, a mucous patch which may appear on any part of the larynx. The patch itself is flat, slightly raised, and surrounded by an inflamed areola; the epithelium may be shed, giving rise to a shallow oval ulcer with smooth base and sharply cut edges.

Symptom.—The only symptom is persistent and painless hoarseness.

Diagnosis and prognosis. The appearance, the recognition of secondary symptoms elsewhere, and the fact that the hoarseness is not amenable to ordinary treatment, generally lead to a correct diagnosis. The prognosis is altogether favourable, and the disease rapidly clears up under antisyphilitic treatment.

The laryngeal lesions produced by **tertiary syphilis** are—(1) infiltrations, which may be diffuse or circumscribed, (2) perichondritis and necrosis, (3) scars and adhesions.

Pathology. (1) *Infiltrations.*—The circumscribed infiltration or *gumma* is not so frequently seen as the diffuse or general gummatous infiltration. The former is usually situated on the epiglottis or on the lateral walls of the larynx; the latter may involve the whole larynx or affect only a part. A laryngeal gumma is smooth in appearance and dull red in colour; it is prone to break down early into a deep punched-out crateriform ulcer with sloughy base and an inflamed areola. The diffuse infiltration presents an irregular, smooth, rounded appearance, is dusky red in colour, and tends to break down into ulcers having the same characteristics as the circumscribed form but spread over a larger area.

(2) *Perichondritis and necrosis.*—Perichondritis may be caused by a gumma over any one of the laryngeal cartilages, the perichondrium becoming involved in the infiltration; or the infiltration may commence in the perichondrium itself. The resulting perichondritis is evidenced

by swelling and impaired movements. Ulceration may follow, with necrosis and exfoliation of the whole or part of the cartilage.

(3) *Scars and adhesions*.—The epiglottis may be partially or wholly destroyed, and in the former case is usually found to be adherent either to the tongue or to the posterior wall on either side. The arytenoids and lateral walls are considerably deformed by thick bands of scar tissue, or a web of cicatricial tissue may be found either uniting the anterior part of the vocal cords, or situated in the subglottic region, leaving only a very small opening for respiration.

Symptoms.—There is produced an alteration in the voice, which may range from a slight hoarseness to complete aphonia, depending on the site of the lesion. As a rule it is hoarse and raucous. Pain is usually present, but is rarely severe and may be absent. Dysphagia is only present if ulceration of the epiglottis has taken place. A certain amount of dyspnoea is generally complained of. It is caused by the perichondritis, the oedema, the fixation of the cords, the formation of cicatrices, and the impaction of exfoliated sequestra. It therefore varies considerably in severity in accordance with the multiplicity of these lesions and their extent.

Complications.—The complications to be feared are septic pneumonia, and the superinfection of tubercle or malignant disease in old-standing cases.

Diagnosis usually presents no difficulty, as, apart from the local changes and appearances described above, confirmation is obtained from the history, the associated lesions elsewhere, the Wassermann reaction, and the results of treatment.

Prognosis depends upon the stage of the disease when it first comes under treatment. In early cases treatment is likely to be successful, with a fair recovery of the voice. In the later stages the voice is invariably altered, and life may be endangered by septic pneumonia, dyspnoea, exhaustion due to necrosis, or hæmorrhage from ulceration. Unless the ulceration is treated early, it is certain to be followed by extensive deformity.

Treatment. Local.—An alkaline spray such as Dobell's solution should be used regularly, followed by insufflation of iodoform or aristol powder. If there is pain, anæsthesin or orthoform powder must be insufflated. Exuberant granulations should be painted with nitrate of silver, 10 gr. to the ounce. If there is oedema,

cold must be applied externally by Leiter's coil, and ice given to the patient to suck; the larynx should be sprayed with a combination of a 2-per-cent. solution of cocaine and 1-in-2,000 adrenalin. If this fails to relieve, scarification or tracheotomy becomes necessary. An impacted sequestrum requires, of course, immediate removal with laryngeal forceps. If symptoms become urgent, laryngotomy or tracheotomy must be performed immediately.

For general treatment, see SYPHILIS.

J. GAY FRENCH.

LARYNX, TUBERCULOSIS OF.—Tuberculosis of the larynx is always secondary to a pulmonary lesion, even when no symptoms in the chest can be detected, and a realization of this must be kept before us as a guide in prognosis and treatment.

Frequency.—The incidence of laryngeal involvement in patients with pulmonary tuberculosis increases with the progress of the latter disease, and so it varies very much in different statistics. Thus the proportion is given clinically as 3 to 25 per cent., whilst post mortem the larynx is seen to be affected in 48 to 52 per cent. of fatal cases.

But a systematic examination of all patients entering a sanatorium, even although these are selected cases, shows that these figures underestimate the proportion. Thus I have found that the larynx was involved in 13·7 per cent. of cases in the first or early stage of consumption, in 27·1 per cent. of those in the second stage, and in 40·8 per cent. of those in the third stage. Of cases which go on to a fatal termination, I doubt if the larynx escapes in more than 25 per cent.

Etiology.—Infection of the larynx from the lungs may take place (a) from the surface of the mucous membrane by the sputum, or (b) from the submucous area where the tubercle bacilli arrive from the lungs by the blood- and lymph-streams. The disease is said to be more frequent in males than in females. It is rarely met with under 10 years of age, is most common between 20 and 40, and diminishes in frequency over 50. Nasal obstruction and chronic catarrh are claimed as predisposing causes, and the bad effects of alcohol, dust, and tobacco are recognized in all laryngeal affections. Syphilis is not uncommonly met with as a complicating factor.

Symptoms.—Subjective symptoms may be absent, especially when the cords are not in-

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involved, and hence the importance of systematically examining the larynx at intervals, in every case of pulmonary tuberculosis. Cough is not an important symptom. Amongst early symptoms are sensations of discomfort and uneasiness in the throat, leading to "hemming" and the feeling of catarrh. Changes in the voice depend on the situation and nature of the local lesion. It may become chronically hoarse and weak if the cords are invaded, but only woolly and muffled if other parts of the larynx are diseased. If stenosis be produced, either by deposit in or below the glottis or by fixation of the cords, dyspnoea and stridor may develop. Dysphagia is common in fatal cases, and is often due to infiltration of the epiglottis. General symptoms are not prominent, and are usually attributable to the condition of the lungs, except in so far as they may be caused by laryngeal pain and dysphagia.

Examination.—The lesions seen in tuberculosis of the larynx vary according to their situation, extent, age, and type. They also vary according to the degree of local and general resistance. They are conveniently studied in the four stages of (1) catarrh and deposit, (2) ulceration and proliferation, (3) perichondritis, and (4) healing—remembering that all may be met with concomitantly. Particular consideration will be bestowed on the early appearances, while the case is in a curable stage; and a briefer account will be given of the advanced lesions which, unfortunately, are generally characteristic of a fatal termination.

The posterior half of the larynx is the region most frequently affected, the order of frequency being generally (1) the arytenoids, (2) the interarytenoid region, (3) the vocal cords, (4) the ventricular bands, (5) the epiglottis. Ulceration is most commonly seen on the vocal cords, and less frequently in the arytenoids.

Early physical signs.—Local pallor, particularly of the palate, is sometimes a striking feature, but it is often present in pulmonary tuberculosis while the larynx remains free. This local anæmia is not constant, and may be replaced by chronic hyperæmia, particularly in men. Laryngeal catarrh, coming on and persisting without any visible cause, with a succulence and loss of tension of the cords, should lead to a still closer scrutiny for any roughness or velvety appearance of the interarytenoid region, thickening or congestion of one arytenoid, of one aryepiglottic fold, or one side of the larynx; for simple catarrh

is generally bilateral and more or less symmetrical, while specific infections are more apt to be unilateral or irregular. Paresis of adduction and tension in the cords may be among the early signs, and a case should never be dismissed as one of functional aphonia, particularly in an anæmic and tired patient, without tuberculosis having been considered.

Interarytenoid region.—This is a favourite area for deposition to take place in the form of a mound or flat mass, which, in favourable cases, may become absorbed, whilst in others it becomes fissured, ulcerates, and breaks down with an uneven, irregular surface. This lesion is often associated with deposition and ulcera-



Fig. 47.—Tuberculosis of the larynx. From a patient who died of pulmonary tuberculosis. The early appearances of laryngeal tuberculosis, viz. infiltration and abrasion of the interarytenoid region and the area above each process vocalis, are seen. (*St. Bartholomew's Hosp. Mus., No. 1613a.*)

tion in the posterior ends of the cords, and in the area just above each vocal process (Fig. 47).

The arytenoids.—A deposit in this region is generally indicated by congestion and a swelling, more or less diffuse, which, owing to the lax submucous tissue, forms readily. The arytenoid eminences are represented as smooth enlargements, which shade off into the aryepiglottic folds, and so are produced the characteristic pear-shaped swellings, which may be red and fleshy, purplish, or an anæmic pink.

Vocal cords.—Tuberculosis of the cords usually takes the form of ulceration, for, owing to the close adhesion of the epithelium to underlying elastic and muscular tissue, a deposit in the limited submucous area cannot increase without breaking down. These ulcers are superficial, irregular, frequently ill-defined, with mouse-nibbled margins and uneven, dirty-grey surfaces. As they progress they become

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surrounded and covered with indolent granulations, sometimes large enough to narrow the airway and embarrass respiration.

Ventricular bands.—Pathological researches show that the ventricle of Morgagni is a favourite haunt of the tubercle bacillus, and, according to some observers, infiltration of the false cords is very common. Clinically, the disease is first noticed along the free margin as an ulcerating deposit or a fringe of pink granulations, which may encroach upon, and lead to apparent narrowing of, the true cords.

The **epiglottis** is not among the most commonly invaded areas and is seldom affected alone. Still, there are cases, in which the type of tuberculosis is not acute, and in which the epiglottis is the first part of the larynx to show a limited, indolent, painless deposit that,

joint is the most common site of perichondritis. Here a deposit may lead to immobility and fixation of the corresponding vocal cord. If both joints be affected and cicatrization take place, the cords may not only be rendered immobile, but the scarring may draw their posterior extremities together, causing stenosis, and even requiring tracheotomy. These favourable developments are rare; more commonly the disease invades the perichondrium by spreading inwards. Perichondritis rarely invades the cricoid cartilage, nor, it is said, the epiglottis. Perichondritis of the thyroid cartilage rarely occurs as an extension of disease, but is occasionally met with as a primary infection.

The trachea.—It is extremely rare for tuberculosis to invade the windpipe before attacking



Fig. 48.—Tuberculoma of the larynx. Phonation brings into view the lower part of the epiglottis, which is characteristically infiltrated and ulcerated.

in appearance, symptoms, and progress, so nearly resembles lupus that it might be distinguished as a "lupoid" form. Apart from this favourable form, tuberculosis of the epiglottis is a serious and distressing complication. In the early stage the patient may only complain of "something in the throat" or of a change in the voice, and it requires a close inspection to detect that the epiglottis has lost, in whole or in part, its shell-like outline, and that the ordinary pink, smooth, glistening surface has in part become thickened, velvety, congested, and deep-red, or that the whole cartilage is swollen to several times its ordinary volume and become a large, rounded, inflamed mass, curved upon itself and standing up above the larynx so as to resemble a turban, a phimosia, a paraphimosis, or a swollen os uteri. Dysphagia is seldom absent in this form and may be agonizing.

Affections of cartilages.—The crico-arytenoid

the larynx, but, when the latter is severely affected, tracheal ulceration is met with, chiefly on the posterior or membranous part of the tube.

In connexion with tuberculosis of the larynx there remain to be considered (1) tuberculous tumours, (2) stenosis, (3) fixation or paralysis of the cords, and (4) adenoma.

1. A *tuberculoma* is a rare manifestation of tuberculosis in the larynx, but a few cases have been recorded in which it has occurred in the form of a small, pale, indolent, and usually sessile tumour. (Fig. 48.)

2. *Stenosis of the larynx* may be brought about (a) by deposit and proliferating granulations, or perichondritis, or (b) by fixation of the vocal cords.

3. *Immobility of a vocal cord* may be the result of (a) plastic infiltration around the arytenoid joint, leading to adhesive perichondritis and spurious ankylosis; (b) ulcera-

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tion followed by scarring and web-formation, or (c) fixation of the cord from pressure on a recurrent laryngeal nerve by a tuberculous thickening of the pleura or a tuberculous bronchial gland.

4. *Œdema of the larynx* is not very frequently met with in tuberculosis, but it may supervene even suddenly on deposits, especially when the tuberculous lesions are near the perichondrium, and in the last stage of phthisis.

Diagnosis.—When viewed with a trained eye, the laryngeal appearances enable a correct diagnosis to be made in the great majority of cases. In those which are doubtful, further help must be sought by a careful investigation of the patient's previous and family history, taking careful note of his weight and daily records of temperature, by a complete examination of the chest including X-ray examination, and a close scrutiny of any sputum for the presence of tubercle bacilli. The reactions with tuberculin are not at present looked upon as of much diagnostic use, but the blood tests by complement-fixation promise to be valuable.

Syphilis is distinguished by its predilection for the epiglottis, the more punched-out character of the ulcer, the more definite deposit, and the Wassermann reaction. The process is more active and less catarrhal. The syphilitic voice is hoarser, but as it is generally painless it is usually raucous and louder. A long-standing history of voice trouble, yet with but slightly impaired general health, is more suggestive of syphilis than of tubercle. Under specific treatment a syphilitic process will improve, but a tuberculous larynx is generally made worse.

The diagnosis from *malignant disease* of the larynx is given on p. 171.

Prognosis.—The invasion of the larynx by tubercle is, next to meningitis, the most serious complication of phthisis. The prognosis has considerably improved with modern methods of treatment, a fair number of slight and early cases recovering spontaneously under sanatorium treatment, whilst in a certain number of selected cases arrest can be brought about by local measures. In acute, progressive, and diffused lesions, with extensive lung mischief, or in patients with loss of weight, quick pulse, fluctuating temperature, poor stamina, a bad family history, or in poor circumstances, the outlook is very gloomy. Not only is a fatal termination more likely, but it arrives sooner, when the larynx is involved.

The most promising lesions are small, in-

tact infiltrations in the interarytenoid region and superficial ulcers on the cords.

Treatment must be based on the recognition of the fact that the laryngeal trouble is but an extension of tuberculosis of the lungs, and that everything which tends to arrest the latter will benefit the throat. The two processes do not necessarily improve *pari passu*, but increased general resistance will strengthen local measures, and it is useless to trouble a patient with these if his phthisis is progressing.

An early case, in a favourable subject, should be sent to a sanatorium, or, when this is impossible, the principles of abundant diet, regular rest and exercise, constant exposure to unvitiated air, and routine medical supervision should be carried out. Tobacco and alcohol must be abandoned. Locally, the most important measure, in slight and early cases, is to secure rest for the larynx; it is best obtained by silence. Even in advanced cases it is well to economize voice-use and to prohibit speaking out of doors.

Sanatorium principles, with more or less silence, are in themselves sufficient to cleanse and soothe a tuberculous larynx, and so do away with the annoyance and irritation of useless sprays, powders, and lozenges. These should be reserved for cases where symptoms call for them: thus, soothing lozenges of carbolic, menthol and liquorice, aniseed and liquorice, heroin, or codeia are ordered as required; alkaline watery sprays, with the addition of a little carbolic acid or menthol, will cleanse local catarrh or suppuration; inhalations of volatile antiseptics, such as the following, may be given from a Burney Yeo inhaler.

Ry Creosot.
Sp. vin. rectific. } partes aequales.
Sp. chlorof.

Six to eight drops on the inhaler every hour.

Ry Acid. carbol. ʒii.
Creosot. ʒii.
Tr. iodi ʒi.
Sp. æth. ʒi.
Sp. chlorof. ʒii.

Six to eight drops on the inhaler every hour.

Powders used for their antiseptic properties are generally more irritating than beneficial, and steam inhalations are useless, except for intercurrent attacks of laryngitis.

Favourable cases with limited lesions which fail to cicatrize can sometimes be healed by galvano-cautery puncture. Thus, of 178 cases

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of laryngeal tuberculosis I found only 36 suitable for this treatment, and of these I obtained a cure in 15 instances. (In 22 other cases out of the 178 the tuberculous laryngitis was arrested spontaneously.) The application can be made under cocaine, by either indirect or direct laryngoscopy. The good effect is brought about, not by destroying the deposit, but by setting up fibrosis around it.

Curettage (like lactic acid) has been largely supplanted by the cautery, but it is sometimes useful to clear away redundant granulations and enable the cautery-point to get access more quickly to its field of action, i.e. the border-line between the deposit and healthy tissue. The large double curette (Fig. 49) is very useful for amputating an infiltrated and ulcerating epiglottis, so as to relieve dysphagia and, occasionally, in hopes of obtaining a cure.

Tracheotomy is seldom performed as a cura-

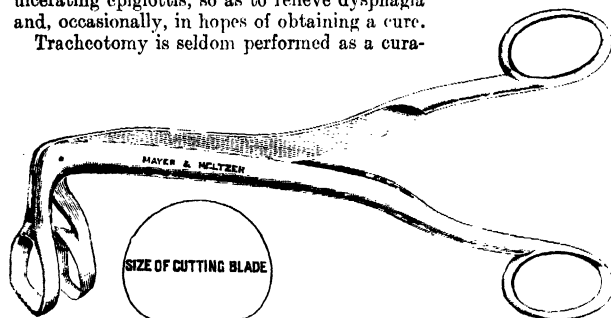


Fig. 49.—Double curette.

tive remedy, but it is sometimes required in cases of increasing stenosis.

Pain and dysphagia frequently subside under vocal rest and suitable soft diet. It may be relieved by getting an assistant to stand behind the patient and make firm pressure at the angles of the jaw at the moment of swallowing. Wolfenden's position may be tried, the patient lying prone on a couch with his face over the end, and sucking the nourishment through a glass tube from a cup on the floor. Or the reverse attitude may prove useful, the patient lying flat on his back and drinking the fluid from a baby's feeding-bottle. Relief may also be obtained by a laryngeal insufflation of 3-5 gr. of orthoform or anæsthesin. It is best given half an hour before the chief meal of the day, and is only effective when there is ulceration. Pain may also be eased by injections of alcohol or novocain into the superior laryngeal nerve. Sprays of cocaine and insufflations

LARYNX, ULCERATION OF

or hypodermic injections of heroin or morphine should be reserved for hopeless cases, when they must not be spared.

Treatment of complications.—If syphilis is coincident it should be energetically treated with salvarsan and mercury. Iodides are not very suitable in tuberculous laryngitis.

Pregnancy is an anxious complication. The induction of abortion or premature labour is not warranted, as the disease advances just as rapidly after the uterus has been emptied. Pregnancy should be avoided in cases of laryngeal tuberculosis, and nursing forbidden.

STCLAIR THOMSON.

LARYNX, ULCERATION OF.—Syphilitic, tuberculous, and malignant ulceration of the larynx are dealt with in preceding articles; here simple ulceration will be considered.

Etiology.—Simple ulceration may be due: (a) To *traumatism*, resulting from intubation, œsophagoscopy, direct laryngoscopy, or from an abrasion or impaction of a foreign body; this form is also seen as the result of swallowing corrosives or boiling fluids. (b) To certain of the infective diseases, such as measles, typhus, typhoid, diphtheria, smallpox, influenza, and broncho-pneumonia.

Pathology.—The traumatic ulcerations result from the infection of simple abrasions. As a rule these are superficial, and tend to heal easily unless the foreign body, such as a fish-bone or other small spicule of bone, becomes impacted, in which case a deeper ulceration may result. In measles, typhus, diphtheria, smallpox, and broncho-pneumonia ulceration is rare. In typhoid it is met with in about 18 per cent. of all cases, and has a tendency to attack the posterior part of the larynx and the edges of the epiglottis. The ulcer is excavated and deep, with a marked area of infiltration, and may lead to perichondritis; it is rarely seen before the end of the third week.

Symptoms.—Husiness or hoarseness of the voice is complained of, and an irritation which causes coughing and some pain; in

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more severe cases there may be stridor, dyspnoea, and dysphagia.

Diagnosis.—Diagnosis is based on the history of the case, the supervention of the laryngeal symptoms indicated above during the course of infective fevers, and the appearance of the larynx.

Prognosis.—In the traumatic type the prognosis is favourable. In the types accompanying the acute infective diseases it is of very serious import.

Treatment.—In traumatic types the foreign body, if one is present, must be removed and an alkaline spray given. In all cases of acute specific fevers, especially in typhoid, a rigid prophylactic régime should be carried out from the beginning. Mouth and teeth should be kept scrupulously clean, and the mouth, pharynx, and larynx sprayed with a 2-per-cent. solution of camphor and menthol in paroleine. When symptoms have supervened the larynx should be sprayed every few hours with an alkaline solution such as Dobell's, and steam inhalations of tr. benzoini co., 1 dr. to a pint of water at 140° F., given. If any signs of dyspnoea or stridor appear, an early tracheotomy should be performed, especially in laryngeal ulceration complicating typhoid fever.

J. GAY FRENCH.

LATERAL CURVATURE (*see* SPINAL CURVATURE).

LATERAL SCLEROSIS, AMYOTROPHIC (*see* MUSCULAR ATROPHY, PROGRESSIVE).

LATERAL SCLEROSIS, PRIMARY.—

This term, which connotes a primary degeneration of the pyramidal tracts, has been very loosely applied to various conditions characterized by spastic paresis of the limbs, in which symptoms other than motor have been slight or absent. But an isolated primary affection of the pyramidal tracts is rare; when these undergo degeneration it is generally in company with a similar change in other tracts, as the dorsal columns in combined sclerosis, or with the spino-cerebellar in addition as in Friedreich's disease, or with atrophy of the ventral horns as in amyotrophic lateral sclerosis.

The most common condition in which disease is limited to the pyramidal fibres is the familial form of spastic diplegia of children; a similar affection is occasionally observed in adults. In these cases the essential pathological lesion is a primary degeneration of the cortico-spinal

fibres, which begins at the distal extremities of the longest, and slowly ascends the cord. Its etiology is doubtful; some cases have developed in families with neuropathic taints, others have been attributed to infection, to exposure, and to syphilis.

Symptomatology.—The onset is very insidious in early or middle adult life. The symptoms are at first only subjective; the patient complains of stiffness of his legs and that they tire too easily. After a variable period his gait becomes obviously affected, his legs move stiffly and awkwardly, his steps grow shorter, and a difficulty in raising his toes from the ground gradually develops, so that he is liable to trip over small obstacles or on rough ground. Examination now reveals considerable spasticity of his legs but little or no diminution in strength. The knee- and ankle-jerks are exaggerated, clonus can be elicited, and the plantar reflexes are of the extensor type. As the disease advances, walking becomes more and more difficult, and may finally be impossible; the stiffness and rigidity of the legs increase, and may become so great that they can scarcely be moved passively. It is always an extensor rigidity—that is, it fixes the hips, knees, and ankles in rigid extension. This naturally impedes voluntary movements, though their strength is as a rule less diminished than would be expected. Reflex spasms are common.

The upper limbs may be affected similarly, but here the symptoms generally develop late and are never so pronounced. The movements of the arms may be merely stiff and awkward. The sphincters often escape till late, but in many cases there is a tendency to constipation and to precipitancy of micturition. In the typical cases there are no sensory symptoms or demonstrable disturbances of sensation, but the severer spasms may produce pains. The disease rarely ascends to the domain of the cranial nerves, but a few cases have been described with symptoms of spastic bulbar disease.

Primary lateral sclerosis is a very slowly progressive and longstanding disease. Some of its victims become ultimately bedridden, but others remain able to get about.

The **differential diagnosis** may be a matter of difficulty, but as primary lateral sclerosis is a slow and very gradually progressive condition, all cases of rapid, subacute, or intermittent course can be excluded. Its possibility may be also neglected in patients

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with obvious sensory symptoms. The greatest danger is its confusion with *disseminated sclerosis*, but the intermittent course of the latter and the presence of ocular symptoms, as diplopia, nystagmus, or symptoms of retro-bulbar neuritis, are generally sufficient to distinguish it. In some cases of *amyotrophic lateral sclerosis* the spastic symptoms precede the appearance of muscular wasting, and in this stage the disease is really a primary lateral sclerosis.

Treatment is unfortunately hopeless, but regular periods of rest and massage tend to reduce the rigidity and give the patient more control over his limbs. Förster's operation of posterior root section may reduce the spasticity of the legs, but the general state of the patient at the stage at which it might be considered rarely warrants it.

GORDON HOLMES.

LATERAL-SINUS THROMBOSIS.—This disease is one of the gravest complications of ear infection, but it is one which, if diagnosed early, frequently yields to prompt surgical intervention. It may appear at any stage in the course of otitis, whether acute or chronic, but it is more usual in acute cases. It gives little or no external evidence of its presence, so that the diagnosis depends almost entirely upon constitutional symptoms, more especially upon the course of the pyrexia.

Pathology.—The septic infection from the ear sets up a localized phlebitis of the sinus, in consequence of which a coagulum forms at and around the infected area. As time goes on, the centre of the clot dissolves into pus, while the phlebitis and clot-formation spread up towards the torcular herophili, down towards the jugular bulb and jugular vein in the neck, and later, in the more chronic cases, as far as the cavernous sinus and the venous sinuses of the opposite side of the cranium. As is usual in septic thrombo-phlebitis, metastatic abscesses are liable to form in the lungs, about the articulations, and elsewhere in the body. The presence of this active septic focus in the circulation is responsible for the symptoms.

A blood-count shows high leucocytosis, and a blood-culture will generally give evidence of the presence of bacteria in the circulation.

Symptomatology.—The symptoms are those of acute septicæmia and pyæmia, with a decidedly intermittent or remittent pyrexia, characterized, as a rule, by rigors.

During an acute suppuration of the middle

ear or an acute exacerbation of a chronic suppuration (*see* OTITIS MEDIA) with the usual local signs and symptoms, the patient has a rigor in which the temperature shoots up from the neighbourhood of 100° F. or lower to 103°, 104°, or 105° F. The rise to this point is rapid, occupying only from two to four hours. The rigor occurs during the ascent. The decline is equally rapid to the former level. During the decline there is profuse perspiration. This swinging temperature, with frequent rigors and perspirations, is characteristic of lateral-sinus thrombosis and continues throughout the whole course of the disease.

If the condition is unrelieved by operation the symptoms gradually assume the characters of severe septicæmia and pyæmia, of which three or four distinct varieties or types are described: (1) the *pulmonary* type, in which the lungs, infected by metastases from the original focus in the lateral sinus, become the seat of pneumonia or broncho-pneumonia; (2) the *typhoid* type, in which diarrhoea and tympanites, combined with the oscillating temperature, produce a broad resemblance to the phenomena of typhoid fever; (3) the *meningeal* type, in which the general constitutional disturbance is combined with the symptoms of septic meningitis; (4) the *pyæmic* type, in which, the case being relatively mild, time is afforded for the deposit and development of metastatic abscesses in various parts of the body, generally around the articulations. As might be expected, these different types will frequently be found to be more or less combined in the same case.

In the earlier stages of the disease, and sometimes throughout its whole course, the patient's senses and intellect are quite clear, and even unilateral headache, though usual, is not invariable. Unless, therefore, the temperature is taken frequently, the occurrence of lateral-sinus thrombosis during an acute ear-infection may be unsuspected until too late. A rigor is of the utmost significance. In children, however, and sometimes also in adults, the characteristic rigors may be absent, and for this reason the temperature should be regarded as the chief guide. In this connexion one may recall the important clinical rule: *In acute otitis media, and in mastoid suppuration, with a temperature over 100° F., look out for lateral-sinus thrombosis.*

Tenderness along the course of the jugular vein in the neck is a late and inconstant symptom of no diagnostic value. I have

LATERAL-SINUS THROMBOSIS

never encountered the "hard cord" in the neck described in most textbooks.

Apart from surgical interference, the duration of the disease varies from a few days in the more virulent to several weeks in the more chronic cases. It is almost invariably fatal unless operated upon.

Diagnosis.—Lateral-sinus thrombosis is mistaken for simple pneumonia or bronchopneumonia, especially in children. An oscillating temperature in *infantile pneumonia*, coupled with a discharge from the ear, should always lead the practitioner to consider the possibility of lateral-sinus thrombosis. In the abdominal type, *typhoid fever* may be suspected and valuable time lost while awaiting the appearance of the Widal reaction. Here, again, the presence of ear disease should lead to suspicions of an infected lateral sinus. The patient's word alone should never be relied upon to exclude middle-ear suppuration. Many people have purulent otitis media without knowing it. Thus in all unexplained pyrexial states the practitioner should himself inspect the ears with mirror and speculum.

Treatment.—No treatment is of any avail save that of operation, and the earlier the operation is undertaken the better. The sinus should be exposed and examined in all cases of ear infection when suspicious oscillations of temperature are observed of over forty-eight hours' duration. To explore the sinus, though not free from peril, is less dangerous than to await the full development of symptoms. Naturally, the careful practitioner will first of all endeavour to exclude such simple pyrexial attacks as tonsillitis, influenza, etc.

In addition to the operation on the sinus and on the jugular vein in the neck, both of which are generally necessary, metastatic abscesses when present will, of course, require local treatment. Many arthritic cases, however, get well without incision, when once the primary focus of infection has been removed.

DAN M'KENZIE.

LEAD NEURITIS (see MULTIPLE NEURITIS).

LEAD POISONING, ACUTE AND CHRONIC (see POISONS AND POISONING).

LEBER'S DISEASE (see OPTIC ATROPHY, FAMILIAL).

LEGG'S DISEASE (see PSEUDO-COXALGIA).

LEISHMAN-DONOVAN BODIES (see KALA-AZAR; ORIENTAL SORE).

LENS, DISLOCATION OF

LENHARTZ TREATMENT (see GASTRIC AND DUODENAL ULCERATION).

LENS, DISLOCATION OF.—Luxation of the lens may be of traumatic or of congenital origin; occasionally it occurs spontaneously in eyes the subject of chronic inflammatory or degenerative processes.

Congenital dislocation is always bilateral; an upward displacement is commonest, and the condition is usually symmetrical in the two eyes. Heredity plays a prominent part in the etiology. The abnormality is detected by illuminating the dilated pupil with the ophthalmoscope, when the equator of the lens appears as a dark crescentic line, and, owing to its prismatic action, two pictures of the optic disc may be seen. The portion of the pupillary area in which there is no lens is, of course, highly hypermetropic, while the other portion is highly myopic. Hence visual acuity is poor, and diplopia may be complained of. Should the lens be small, as is not infrequently the case, the anterior chamber may be deep and the iris tremulous.

Treatment. If the edge of the lens passes across the undilated pupil one may attempt to obtain improved vision by correcting either the myopic or the hypermetropic error—which ever gives the better result. It is sometimes advisable to enlarge the pupil, either by nicking its margin, or by performing a small iridectomy. An attempt to get rid of the lens itself is to be undertaken only with great circumspection; needling is difficult, and the operation is often badly borne. Sometimes in later life a partial luxation becomes complete, so that the pupillary area is cleared.

Traumatic dislocation results usually from a blow with a blunt body. It may take place forwards into the anterior chamber, or backwards into the vitreous; or it may be partial, in which case one edge is usually canted forwards. When dislocated anteriorly the lens is visible (sometimes with difficulty if its transparency be perfectly preserved), lying in the anterior chamber, which is very deep. Glaucoma results both from direct interference with drainage and because the iris, contracting behind the lens, shuts off communication between the posterior and anterior chambers. By the ophthalmoscope a lens dislocated into the vitreous may be detected lying on the ciliary body below. The anterior chamber is moder-

ately deepened, the iris tremulous, and the pupil very black. In this case, also, glaucoma frequently results, probably owing to a forward displacement of the vitreous. In partial dislocations there are shallowness of the chamber on one side, and increased depth, frequently with tremulous iris, on the other. Glaucoma may result from occlusion of the corneo-iridic angle owing to direct pressure on the iris root.

Treatment.—All these conditions are grave, but in some instances the lens can be removed with success.

GEORGE COATS.

LENS, EXAMINATION OF (see EYE, EXAMINATION OF).

LENTICULAR DEGENERATION, PROGRESSIVE.—A rare nervous disease, though one of great interest because of the constant association of disease of a certain part of the brain with disease of the liver and, to a less extent, of certain other internal organs.

Etiology.—The cause is at present unknown, but the evidence goes to show that some toxin is generated or elaborated in the alimentary tract or in the liver, and that this has a specific action on the lenticular nucleus. The disease occurs mostly in young people and has frequently been observed to be familial.

The clinical **symptoms** are of gradual onset and essentially progressive. They consist mainly of bilateral involuntary movements of the limbs, nearly always of the nature of tremor, bilateral spasticity of the limb musculature, dysarthria, dysphagia, muscular weakness, progressive emaciation, and contractures. With these may be associated emotionalism and certain symptoms of a mental nature. As far as is known, the disease, after a longer or shorter period, is fatal.

Pathologically it is characterized predominantly by bilateral symmetrical degeneration of the corpus striatum, in particular the lenticular nucleus, which may be so disintegrated as to lead to cavity-formation; and by cirrhosis of the liver, which, though constant, rarely gives rise to symptoms.

Treatment.—The writer has tried the administration of urotropine over a long period, without, however, producing any obvious improvement. Treatment must remain empirical until the nature of the disease is elucidated.

S. A. KINNIER WILSON.

LENTIGO.—This term is used both for the ordinary freckle due to exposure to the sun, and for the "cold" freckle which arises on covered parts and is probably a pigmentary naevus. Both are forms of pigmented macule due to the excessive deposit of melanin in the skin. As a rule no treatment is indicated, but, if considered necessary, a bleaching process with hydrogen peroxide (10 vols.) may be carried out, or if the spots are few they may be touched with pure carbolic acid. The latter measure demands caution, or disfiguring scars may be caused. The wearing of brown veils, or the use of lotions or ointments containing quinine, is a preventive.

H. MACCORMAC.

LEONTIASIS OSSEA (*syn.* Hyperostosis Cranii).—A rare form of enlargement of the cranial bones ascribed to osteo-sclerosis. All the bones of the head are affected and form prominent bosses. The cranial vault, especially the forehead, increases in size, and a leonine aspect may be acquired. The thickening of the bones leads to reduction of the orbits, to constriction of the foramina, and to partial or complete obliteration of the air sinuses. Exophthalmos and displacement downwards of the eyeball follow. Compression of nerves in the narrowed foramina causes neuralgia, cranial paralysis, headaches, blindness or deafness; and compression of blood-vessels leads to mental disturbance and engorgement of tributary veins. The condition may start in childhood, but is regarded by some as a variety of osteitis deformans. It may persist for several years. Its cause is unknown. Treatment is of little avail, though operative procedures may afford relief.

FREDERICK LANGMEAD.

LEPROSY.—A general disease with a chronic and paroxysmal course, characterized by lesions in the skin, mucous membranes, nerves, and viscera.

Although leprosy has been known from very ancient times, and during the last half-century has been the subject of the closest observation and scientific study, there is much that still remains unknown about it, and there is no disease which, in the present state of our knowledge, may be said to be more mysterious. The duration of the incubation period is indefinite; the invasion symptoms are not characteristic; the causal bacillus has neither been cultivated nor successfully inoculated in lower animals, and the path of its infection and its manner of transmission from man to

man are uncertain; the reason why in one case the bacilli become widely disseminated in the connective tissue of the body, causing lepromata not only in the skin but in almost every organ, while in another case they are confined for an indefinite period to the nerve-sheaths, is unexplained; and, with the exception of the improvement which may follow treatment by certain vegetable oils, the disease is slowly progressive, and the old saying, "Once a leper always a leper," unfortunately in the great majority of cases still remains true.

Etiology.—The disease is caused by the *Bacillus lepræ* (PLATE 5, Fig. 2, Vol. I, facing p. 148), a rod-shaped acid-fast bacterium closely resembling the tubercle bacillus, but differing in that, while the tubercle bacillus can be cultivated on artificial media and successfully inoculated in certain lower animals, all attempts to cultivate the lepra bacillus have failed, and no animal has been found to be susceptible to it. There is a disease in rats known as rat-leprosy which is due to an acid-fast bacillus, but it is a different disease, and not human leprosy in the rat.

Leprosy is transmissible from man to man, but the method of transmission is unknown: it may be by direct contact or it may take place indirectly by means of feeding-utensils, towels, etc., or through the medium of some biting insect; flies, mosquitoes, lice, and bugs have all been suggested as possible carriers. The path of invasion is also uncertain: it might be through the skin, alimentary tract, respiratory passages, or genital organs; there are cases on record which point to each of these paths—e.g. a medical man became inoculated through a wound in the finger while attending a leper woman in labour, and there are authenticated instances in which the virus appears to have been introduced through vaccination. Many articles of diet have been held responsible, such as rice, pork, fish, etc., and of these the one which has attracted most attention is fish, especially dried, decomposed fish. This theory, however, is not generally accepted, and, without going into the arguments for or against it, it is sufficient to point out that it is not corroborated by any scientific evidence, and that the lepra bacillus has never yet been found in decayed fish.

The frequency with which nasal symptoms, such as rhinitis and epistaxis, occur as early manifestations of leprosy, and the comparative ease with which the bacilli may be found in the

nasal discharge, have suggested the possibility of the upper respiratory tract being a path of infection. So far, bacilli have not been found in the air; consequently they are not likely to have reached the nose by inhalation, and should infection take place in the nose the most likely manner would be by inoculation through an abrasion in the mucosa caused by "picking."

With regard to the genital organs, if infection takes place through them it must be the result of the presence of some sore such as lepromatous ulcer, as the uterine discharges and the semen do not contain the bacilli unless secondarily contaminated.

The contagiousness of leprosy is now generally accepted, but it is found to be comparatively slight, as is shown by the rarity of matrimonial infection. At the Second International Conference on Leprosy, at Bergen in 1909, the general opinion on this matter was embodied in the following resolution: "Leprosy is a disease which is contagious from person to person, whatever may be the method by which this is effected, and every country is within the range of possible infection by the disease."

Several attempts have been made to transmit it from an infected to a healthy person, but with negative results, as in Arning's inconclusive experiment in which a criminal was inoculated on the arm with a lepromatous nodule. There are numerous instances on record, however, in which persons previously healthy have acquired it by going to a country where it is endemic and living in close association with lepers. And there are two instances which have come under the observation of the writer in which boys have become infected with leprosy in Great Britain by living in one case with lepromatous parents and in the other with a lepromatous brother.

Predisposing causes.—The spread of leprosy is undoubtedly favoured by *unhygienic surroundings*, as it occurs chiefly in overcrowded and insanitary districts, and in association with poverty and malnutrition.

Sex.—Men seem to be more often attacked than women, in the proportion of 3 to 2.

Age.—The disease does not occur in infancy, but it appears most frequently in childhood, partly from the delicacy of tissues in children and partly from their close contact with lepromatous parents or attendants. It rarely begins after middle life.

Heredity.—There is no evidence of congenital leprosy, and neither the lepra bacillus nor any

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lesion suggestive of its presence has been detected in the stillborn children of lepers.

Symptomatology. Incubation period.—The duration of the incubation period is unknown. In some cases only a few weeks seem to have elapsed between the presumable time of infection and the development of symptoms, but in the majority of cases it has appeared to be years. Five or six years has been suggested as an average period.

Invasion.—In some cases the disease develops insidiously, its first sign being some cutaneous lesion; in other cases the invasion is heralded by prodromal symptoms suggesting a general intoxication, such as febrile attacks, vomiting, pains in the back and joints, epistaxis, and frontal headache, which may precede the eruption by a few weeks or may be present for years before it appears.

Varieties.—After the prodromal phase, local disorders develop, in the form either of definite cutaneous lesions, such as nodules or plaques, or of peculiar sensations over certain discoloured areas, such as hyperæsthesia, pricking and itching. On this account two varieties of leprosy have been distinguished, namely, nodular leprosy (*lepra tuberosa*) and nerve leprosy (*lepra maculo-anæsthetica*). Cases of mixed leprosy also occur in which both types of lesions are present. This division, though useful for descriptive purposes, is purely arbitrary, as in cases which begin as nodular leprosy maculo-anæsthetic lesions may develop eventually, while nodular lesions may be superimposed in maculo-anæsthetic cases.

Nodular leprosy.—The first sign of nodular leprosy is the appearance of erythematous patches, accompanied by a rise in temperature; they either disappear when the temperature declines, or become raised into nodules or plaques from a cellular infiltration of the deeper parts of the skin. New nodules (*lepromata*) tend to come out at irregular intervals, either singly or in crops, and each outbreak is accompanied by a rise in temperature (leprotic fever). The nodules have a smooth surface, are round or irregular in shape, vary in size from a pea to a walnut, and in white skins have a dusky-red colour. They may occur anywhere on the skin, but are most common on the face, especially about the forehead, cheeks, nose, lobes of the ears, and on the forearms and thighs. In advanced cases they may be so numerous as to involve practically the whole skin of the face. The hairs fall out wherever they occur, and the eyebrows and eyelashes

are generally lost, but the hair of the scalp, as a rule, is unaffected, *lepromata* being rare in that situation. As the case progresses the forehead becomes irregularly thickened, studded with nodules, and broken up by horizontal fissures; the supraciliary ridges become prominent, the nose thickened and coarse, the lips everted, the lobes of the ears swollen, and a heavy appearance is produced to which the old name *leontiasis* is singularly appropriate.

The *lepromata* may become hard, fibrous, and persistent, or they may break down like superficial gummata, forming ulcers with an irregular border and a base covered with a glairy, sero-purulent discharge. Either spontaneously or under treatment the ulcers may heal, being replaced by hypertrophic scars and leading to contractions and disfigurement.

The glands are usually affected, especially those of the neck, throat, axillæ, and groins, and may become so large as to interfere with the movements of the joints, or to impede swallowing.

The eyes are usually attacked and become suffused and painful. Small *lepromata* or a diffuse leprous infiltration may appear on the conjunctiva and cornea, or the disease may extend to the anterior chamber and lead to iritis or irido-cyclitis. The eye becomes swollen at first, and, when the inflammation subsides, contraction and blindness may supervene. The eyelids are liable to attack, and this may lead to cicatricial changes, ectropion, exposure of the bulb to septic infection, and even destruction of the eye.

The mucous membranes are generally invaded, and *lepromata* are frequent in the nose, blocking the air-passages, or involving the cartilage and, by breaking down, causing flattening. When present in the throat they are apt to interfere with mastication and deglutition and, when they appear on the epiglottis and vocal cords, to cause a hoarse voice.

The nails are sometimes affected, and may present ridges or furrows, or become thickened, brittle, and opaque.

In children the development of the genital organs is retarded. Menstruation is delayed or prevented in the female, and imperfect development of the testicles in the male may cause impotence.

The changes in the blood and the urine are not characteristic.

As the disease advances, the face becomes more and more deformed. The eyes are destroyed, and the sense of smell and taste may

be lost. Through the difficulty of swallowing, the patient becomes weaker and weaker. Internal organs such as the liver and spleen may be implicated, and derangements of the digestion and diarrhoea may supervene. In most cases the brain is not affected, but the prostration gradually increases, and the patient dies in a state of inanition or from some intercurrent disease of the lungs or kidneys.

Maculo-anæsthetic or nerve leprosy.—In this form of the disease the bacilli invade the nerve-sheaths and set up a lepromatous hyperplasia, the pressure of which on the nerve-fibres causes at first local irritative symptoms, and later anæsthetic and trophic lesions. In the irritative phase the area of distribution of the affected nerve is the site of sensory disturbances such as hyperæsthesia, shooting pains, twitching of the muscles, flushing, and occasionally of vesicles or small bullæ, followed by the appearance of erythematous or pigmented macules or patches, slightly raised, smooth or scaly, which tend to become involuted in the centre, giving rise to circinate lesions with slightly raised scaly borders. In the centre of the patches the hair is lost, the sweat-glands atrophy, and the skin becomes wrinkled. The coalescence of contiguous patches results in the formation of large areas enclosed by an irregular gyrate border. The patches are insensible to heat, cold, or pain, so much so that a burn may occur on them without the cognizance of the patient. The peripheral nerves most commonly involved are the ulnar, peroneal, sciatic, and median, and the affected nerve may be uniformly thickened but more often presents fusiform swellings. In the atrophic phase, changes supervene in the muscles with a loss of muscular power, especially noticeable on the face and extremities. The muscles of the eyelids are frequently involved, leading to ectropion and rendering the eye liable to injury and septic infection. Paresis of the facial muscle causes distortion and a staring, expressionless look. Paralysis of the extensors of the fingers is responsible for "main-en-griffe" or leper-claw, and the involvement of the antero-external muscles of the leg causes foot-drop. Trophic changes such as ulceration occur in the skin and deeper tissues and may lead to necrosis and absorption of the bone, and to the loss by spontaneous amputation of fingers, toes, or even larger portions of the limbs.

This type of leprosy runs a longer course than the nodular, and when death supervenes it is frequently the result of some intercurrent

disease such as tuberculosis or amyloid degeneration of the kidneys.

Histopathology.—The bacilli are disseminated through the system either by the lymph-stream or by the blood. On reaching the connective tissue they set up a mild form of inflammatory reaction which results in the production of a leproma. Wherever situated, lepromata have the same structure, which consists of a loose, delicate fibrous stroma enclosing innumerable colonies of bacilli in clusters or globi, with here and there a few plasma-cells and an occasional giant-cell.

In the maculo-anæsthetic cases the cutaneous lesions are not due to the presence of bacilli in the skin, but are caused indirectly by the pressure of the lepromatous infiltration on the nerve-fibres.

Diagnosis.—The diagnosis of leprosy presents no difficulty, except in its early stages. In nodular cases it has to be made chiefly from syphilis and tuberculosis. In *syphilis* the rapidity with which the symptoms disappear under mercury and salvarsan, which have no effect on leprosy, serves to distinguish them; in *tuberculosis* the cutaneous lesions are usually confined to certain areas, while in leprosy they are widely distributed. In either case a diagnosis can easily be established by demonstrating the lepra bacilli; this is done by scratching the surface of a leproma, squeezing the serum on a slide, and staining it by the Ziehl-Neelsen method.

The maculo-anæsthetic patches may simulate *erythema multiforme*, but the lesions of leprosy are more lasting and are associated with derangements of sensibility. Occasionally they may suggest patches of *ringworm*, but the anæsthesia and the absence of fungus serve to differentiate them. Advanced cases of nerve leprosy may have to be distinguished from *syringomyelia*, but in the latter disease cutaneous lesions are absent, and the affection is of central origin and often unilateral.

Prognosis.—The outlook in leprosy is invariably bad, and, however quiescent the disease may become, it is impossible to assert that it has been completely eradicated. In nodular leprosy the patient may live on an average twenty years, while in maculo-anæsthetic cases the average is thirty years or more.

Prevention.—The only possible way to prevent the spread of leprosy is by isolating the leper, and it has been found that wherever proper measures of segregation have been introduced the disease has steadily declined.

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The ideal condition is segregation in a medically supervised institution, or, failing that, at home with proper attention and subject to regular medical inspection. Obviously, nodular cases with open sores or profuse nasal discharge are the most dangerous, but in the present state of knowledge it is unwise to assert that close association with the disease in the maculo-anæsthetic form is absolutely safe. As a preliminary to any form of segregation, it is essential that leprosy should be made notifiable. Children born of lepers should be separated from their parents as soon as possible and kept under observation. Lepers should on no account be allowed to follow any occupation where they have to sell or handle foodstuffs, and all clothing which has been worn by them should be disinfected.

Treatment.—Although no absolute cure has yet been found for leprosy, much can be done in the way of palliation. It is difficult to determine the value of treatment, as the disease has a natural tendency to spontaneous remissions, which may last for years, and any drug used at the time is apt to get the credit for the improvement; consequently, in estimating the value of any treatment, it is essential that the remedy should have been employed in a considerable number of cases and over a long period.

Internal treatment.—Of the many remedies which have been advocated for leprosy, those that appear to have been most uniformly beneficial have been certain vegetable oils, of which the best-known is chaulmoogra oil, obtained from the seeds of *Taraktogenos kurzii*. This may be given either by the mouth or by injection. By the mouth it is best taken in capsules, beginning with 5-10 m. t.d.s., increased according to the toleration of the patient. Unfortunately it is liable to cause digestive troubles, but these may be minimized by combining the oil with malt. The oil may be given intramuscularly with camphorated oil, but this is a painful procedure, and a better method is to inject it in the form either of colloidal chaulmoogra (Crookes) or of gynecardate of soda, a derivative of chaulmoogra; if the latter is used, 2 gr. in 1 c.c. of sterilized water, with $\frac{1}{2}$ per cent. of carbolic acid added, are injected twice a week subcutaneously, or $\frac{1}{2}$ c.c. of a 3-per-cent. solution intravenously. As a result of the injections a reaction occurs which is both general and local, and is said to be accompanied by a destruction of the bacilli, but in this disease the mere occurrence of the local reaction does not prove the drug to be a specific.

LEUCOCYTOSIS AND LEUCOPENIA

Recently Sir Leonard Rogers has recommended the injection of gynecardate of soda intravenously, and MacDonald and Dean have claimed benefit from injections of ethyl esters of the fatty acids of chaulmoogra oil.

Other remedies have been tried, such as ichthyol, iodide of potassium, mercury, and salvarsan, but no lasting benefit has resulted from them; and various serums, vaccines, and antitoxins have given equally uncertain results. Until the bacillus has been cultivated it is impossible to prepare a "leprolin" to correspond with tuberculin, and the use of antileptotic serums prepared on similar lines to antiphtheritic serums is unattended by permanent benefit.

Apart from specific treatment, general treatment is of the utmost value both in staying the progress of the disease and rendering the life of the patient more bearable. Tonics such as cod-liver oil, strychnine, and arsenic improve the general condition and increase the resistance to the bacillus, and any definite symptom should be dealt with on general medical principles. Sanatorium treatment, with plenty of fresh air and good food, is of as great value in leprosy as in tuberculosis.

Local treatment.—The patient should be kept scrupulously clean by daily baths, to which boric acid or some suitable antiseptic should be added when suppurating sores are present. Ulcers should be dressed frequently, and nasal discharges, ulceration in the mouth and throat, conjunctivitis and keratitis suitably treated. When any gangrene appears about the extremities, surgical intervention is advisable.

J. M. H. MACLEOD.

LEPTOMENINGITIS (see MENINGITIS).

LEPTUS AUTUMNALIS (see HARVEST-BUG RASH).

LEUCOCYTHÆMIA (see LEUKÆMIA).

LEUCOCYTOSIS AND LEUCOPENIA.—

Leucocytosis is a reactive and purposeful increase of the normal hamie leucocytes. Although the normal adult count is about 7,000 per c.mm., counts below 10,000 are not usually regarded as leucocytosis. The diagnosis from leukæmia, which has been described as a purposeless and wanton increase in the leucocytes, formerly depended solely on the number of leucocytes, and it is true that a white count of 100,000 is extremely rare except in leukæmia. On the other hand, in

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aleukæmic phases of leukæmia the total white count may not be above the normal. The distinction lies in the nature of the cells: myeloid leukæmia is characterized by the presence of myelocytes or of myeloblasts (non-granular myelocytes, promyelocytes, lymphoid-cytes); lymphoid leukæmia by lymphocytes, or at least mononuclear cells, in preponderating numbers, commonly over 90 per cent. of the total white cells. Leucocytosis consists in an increase of the normal hæmic leucocytes, and mainly of the polymorphonuclears, which make up 80 to 95 per cent. of the total increase. A very high polymorphonuclear count is in favour of suppuration.

Leucocytosis has been divided into: (1) Physiological, occurring in infancy, pregnancy, and after exercise, massage, hot and cold baths, and during digestion; in some of these circumstances the leucocytosis is slight and due to displacement of leucocytes. (2) Pathological, which occurs in (a) most infective conditions, being more prominent in coccic than in bacillary invasions; the main exceptions are typhoid and paratyphoid fever, influenza, malaria, measles, rubella, mumps, most cases of uncomplicated tuberculosis, some cases of sub-acute bacterial endocarditis, leprosy, Mediterranean fever, kala-azar; (b) toxic states, such as diabetic coma and uræmia, after some drugs (e.g. digitalis and cinchamic acid), and in the gouty paroxysm; (c) after hæmorrhage; (d) in some cases of malignant disease; (e) shortly before death.

A leucocytosis of 30,000 in appendicitis suggests suppuration, and a lower count which steadily increases has the same significance, whereas a falling count is a good sign. Absence of leucocytosis may be due to a highly virulent infection paralysing the bone-marrow and preventing any reaction. In scarlet fever there is a leucocytosis, and thus a diagnosis may be made from a doubtful case of measles. The detection of leucocytosis in diseases usually not showing it—for example, enteric fever—points to some complication. According to Arneft, tuberculosis leads to a relative increase in the number of the immature polymorphonuclears, namely, those with a one- or a two-lobed nucleus.

Lymphocytosis, or excess of the hæmic lymphocytes, occurs physiologically in infants. It is seen, sometimes in a high degree, in whooping-cough. It is more likely to be met with in bacillary than in coccic infection, but exceptional cases of infective wounds, boils, and

widespread streptococcic adenitis of tonsillar origin show a lymphocytosis which is distinguished from lymphoid leukæmia by recognition of the infective focus, by the slighter lymphocytosis, and by its course (Cabot); in adults this infective mononucleosis shows a number of pathological forms, all probably derived from lymphoid tissues. Secondary syphilis, juvenile gastro-enteritis, rickets, lymphatism, Graves's disease, longstanding pernicious anæmia, influenza, the æbriile periods of trench fever, and irritation of lymphatic glands cause relative increase in the lymphocyte count. Lymphocytosis may also occur after splenectomy, sometimes in splenic anæmia, and in early lymphadenoma. An increase in the large mononuclears is in favour of a protozoan infection.

Eosinophilia occurs in many cases of invasion by animal parasites, for example trichiniasis, bilharziasis (schistosomiasis), and intestinal worms, especially ankylostomiasis. But in hydatid disease eosinophilia is inconstant and a negative count is of no value. In some skin diseases, more particularly pemphigus and dermatitis herpetiformis, there may be a high but transitory eosinophilia. It occurs in asthma, and here it may be mentioned that the occurrence of Charcot-Leyden crystals appears to be connected with eosinophilia. Some eosinophilia may occur after splenectomy, injection of serums and vaccines, such as tuberculin, in malignant disease, especially sarcoma of bone, in ovarian cysts, and exceptionally in lymphadenoma. Some drugs, either with or without a skin eruption, may cause an eosinophilia. In myeloid leukæmia there is an enormous absolute, though little or no relative, increase in the eosinophils.

Myelocytosis.—When the bone-marrow is stimulated some myelocytes escape into the blood-stream (stimulation myelocytosis), but not in such numbers as to compare with myeloid leukæmia. Thus a few may be found in leucocytosis, septicæmia, pernicious anæmia, the anæmias of children, erythræmia, diphtheria (especially in fatal cases), and sarcoma of bone. Acute myelæmia has been described in exceptional cases of milary tuberculosis.

Leucopenia, or diminution of the leucocytes in the blood-stream, may be due (1) to failure of the bone-marrow to respond, for example, in very severe infections and in aplastic anæmia, especially that due to benzol poisoning; (2) possibly to excessive destruction of leucocytes; or (3) to accumulation in the blood-

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vessels of the viscera, for example the initial leucopenia in anaphylactic shock, in which the pulmonary vessels are crowded with leucocytes. It may sometimes be difficult to decide which of these factors is at work, but the first is the more important. The term leucopenia is usually employed when the white count falls below 5,000 per c.mm., as in enteric fever, malaria, influenza, pernicious *anæmia*, chlorosis, starvation, Gaucher's disease, and in some cases of tuberculous adenitis. Other conditions in which leucocytosis does not occur and in which leucopenia may be observed are mentioned under Leucocytosis.

H. D. ROLLESTON.

LEUCODERMIA.—An abnormal condition of pigmentation of a non-congenital nature characterized by the appearance of white patches with convex borders gradually invading the surrounding skin.

Etiology and pathology. Very little is known of the processes which modify the pigmentation of the skin. In the condition under consideration the dyschromia may be primary, or may occur as a secondary phenomenon following some other skin disease. Many authorities hold that a toxæmia is responsible for the abnormality. In the white areas pigment is absent, an observation commonly accepted. Whether the surrounding skin is hyperpigmented or not is a point upon which opinion is not unanimous.

Diagnosis.—As a rule diagnosis is not difficult, but care should be taken to distinguish between leucoderma and *chloasma*—in which the convex border is made up from the pigmented area. The various conditions forming *scar tissue* are easily distinguished by the presence of atrophy. Occasionally slight difficulty may be encountered in tropical countries where *leprosy* is endemic, but in such cases the *anæsthesia* of the skin over the white patches affords a reliable guide. A characteristic but uncommon variety of leucoderma is occasionally met with in *secondary syphilis* as a dappled appearance on the neck, the lesions in this case being small, about the size of a sixpence.

Treatment is most unsatisfactory, because although it is possible to cause pigment to form in the depigmented areas, as by X-ray irradiation, such measures increase the pigmentation in the surrounding zone. The application of suitable cosmetics is the only satisfactory means of disguising the unsightly appearance.

H. MACCORMAC.

LEUCONYCHIA (*see* NAILS, DISEASES OF).

LEUCOPENIA (*see* LEUCOCYTOSIS AND LEUCOPENIA).

LEUCOPLAKIA LINGUÆ (*see* TONGUE, SYPHILIS OF).

LEUCOPLAKIC VULVITIS (*see* VULVA, DISEASES OF).

LEUCORRHEA.—Normally the vagina is kept moist by secretion from the cervical glands and by that from the vaginal walls and the endometrium. The mixed fluid is colourless or slightly yellow and varies greatly in amount. The term "*leucorrhœa*" denotes that it is excessive.

"*Leucorrhœa*" means literally a white discharge, but the term should be restricted to cases in which the secretions are simply increased, and not applied to purulent or infective discharges, or to the watery discharge frequently seen in cases of carcinoma of the cervix.

Most of the vaginal fluid comes from the glands of the cervix, which secrete a viscid mucoid material resembling uncooked white of egg. In cases of *leucorrhœa* this fluid can be seen coming from the external os; it is usually turbid when it collects in the vagina.

Etiology.—Any condition causing congestion, overgrowth, or inflammation of the glands renders the secretion excessive and gives rise to *leucorrhœa*.

The result of congestion is seen in the *leucorrhœa* of pregnancy, in that just before and just after the periods, and in that due to subinvolution and retroversion of the uterus. *Leucorrhœa* due to overgrowth appears in cases of adenoma of the endometrium, in old lacerations of the cervix in which there is an increased area of glandular epithelium, in erosions and mucous polypi. It occurs as the result of inflammation in endometritis, cervical catarrh, and old gonorrhœal infection of the cervix.

In many cases there is no obvious local cause, the presence or absence of *leucorrhœa* seeming to depend upon the general state of health. It is frequently met with in young girls, especially those who are *anæmic* or overgrown, about the time of puberty and for some years subsequently.

The only symptom complained of in many cases is the presence of the discharge. Pa

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tients, however, frequently complain of back-ache and of feeling poorly and tired, symptoms probably due as much to the cause of the leucorrhœa as to the leucorrhœa itself. Considerable irritation is uncommon, although in severe cases chafing of the skin around the vulva may be troublesome on account of the moisture of the parts and the constant necessity for wearing a pad.

Leucorrhœa may, however, cause sterility.

Diagnosis.—Before making a diagnosis of leucorrhœa it is necessary to ascertain that the discharge is colourless and not purulent.

If the patient is a young unmarried girl and a reliable account of the character of the discharge can be obtained, local examination should be avoided, unless the discharge is unusually great or persistent, when an examination under an anæsthetic should be advised.

In ordinary cases the vulva should be inspected and the vagina examined through a speculum. If there is no pronounced inflammation of the vulva or vaginal walls and the discharge is not offensive, purulent, or blood-stained, the case may be regarded as one of leucorrhœa.

Treatment and prognosis.—In cases in which the health is poor and no local cause for the discharge is found, treatment should aim at improving the patient's general condition. Young anæmic girls who are growing quickly should not be pressed to work hard at school, but should be out of doors as much as possible and lead regular, healthy lives. Iron and other tonics should be given. In all cases it is important to remember that the patient's state of health has a marked influence on the condition, and should always be considered in deciding upon the treatment.

In the majority of cases local treatment is required, and must vary with the cause of the leucorrhœa. When the excessive secretion is due to cervical catarrh it may be most troublesome and intractable, but various substances applied to the cervical mucous membrane are valuable in checking the excessive secretion. The cervix should be exposed through a Ferguson's speculum, and all discharge carefully wiped away from the upper part of the vagina and cervical canal with cotton-wool on a Playfair's probe. Applications can then be brought into contact with the cervical mucous membrane. Those found to be most useful are pure carbolic acid, copper sulphate

(8 per cent.), iodized phenol and strong solutions of iodine. The fluid selected should be applied with wool on a Playfair's probe and introduced as far as possible into the canal, care being taken to remove all excess before withdrawing the speculum. This process should be repeated, if necessary, at weekly intervals for three or four weeks, and between the applications daily douching with two or three pints of some warm unirritating solution such as boric acid, alum, zinc sulphate, or common salt, each in the strength of 1 dr. to the pint, should be ordered. Douches are useful in washing away the discharge, and may be valuable in reducing the inflammation in the cervix, but cannot reach the cervical mucous membrane. Strong antiseptic douches are valueless, often do harm by irritating the vaginal walls, and should be avoided.

If these methods fail, more prolonged action of drugs can be obtained by applying them in the form of vaginal tampons in contact with the cervix. Glycerin, ichthyol, iodine, or silver salts may be applied in this way. A tampon should be introduced as far as the cervix on each of about ten successive days, and removed next morning; before introduction and after removal a large mild douche should be given.

If this method fails also, operation must be undertaken. Both the cervix and the uterine cavity should be curetted thoroughly, and strong iodine or carbolic acid applied, for it is impossible to be sure that the endometrium of the uterine body is healthy and is not the source of some of the discharge.

Sometimes discharge may persist in spite of curetting, and excision of the cervical mucous membrane or amputation of the cervix may be required. Where mucous polypi are found, their removal, with curetting and swabbing the cervix, should cure the discharge.

With old lacerations of the cervix and erosions the discharge may be greatly reduced by swabbing the glandular area with carbolic acid, but repair of the cervix in the one case and curetting in the other may be needed. In cases due to retroversion the position of the uterus should be corrected.

Curetting is necessary when endometritis or adenoma of the endometrium exists, and probably also in cases of subinvolution, although, in the latter, treatment by glycerin tampons may be beneficial.

J. P. HEDLEY.

LEUKÆMIA

LEUKÆMIA (*syn.* Leucocythæmia).—The cause of leukæmia, though much debated, is unknown. Parasites have been described by Löwit (an intracorpuscular protozoon), White and Proescher (a spirochæte), Steele, Simon and Judd (a diphtheroid organism), Auer (intracorpuscular crescents, rods, and granules), and Ellermann (a filterable virus), but confirmation is lacking. The clinical features, especially of acute leukæmia, suggest an infective origin, but there is no proof of this, though hæmic infection may occur as a secondary process, particularly in acute leukæmia, and some cases of streptococcic infection may show an enormous lymphocytosis. The opinion that leukæmia is allied to, if not a form of, sarcoma involving the leucoblastic tissues, especially the bone-marrow, is difficult to disprove. Formerly the myeloid and lymphoid forms of leukæmia were thought to be different diseases, but recent investigation shows that they are closely allied, that they depend mainly on proliferative changes in the bone-marrow, and that the difference in the hæmic cells depends in great measure on the stage in the evolution of the primitive marrow-cell at which the riotous proliferation occurs. For convenience, acute and chronic forms of myeloid and lymphoid leukæmia, and chloroma, will be described separately.

CHRONIC MYELOID LEUKÆMIA (*syn.* Myelogenous, Myelocytic, or Splenomedullary Leukæmia, or Leucocythæmia; Myelæmia)

This condition is not common. It usually occurs between 20 and 50 years of age, more often in males, and is very rare in children. The cause is quite unknown; injury, pregnancy, and malaria cannot seriously be incriminated.

Morbid anatomy.—The bone-marrow is usually of a pale-red colour, but may be pale-yellow (pyoid), and occasionally it may have a greenish tint as in chloroma. The hyperplastic marrow extends and takes the place of the fat in the shafts of the long bones. Microscopically the marrow shows hyperplasia of much the same cellular elements as in normal marrow, the colourless cells, especially those of the granular series, predominating (Muir).

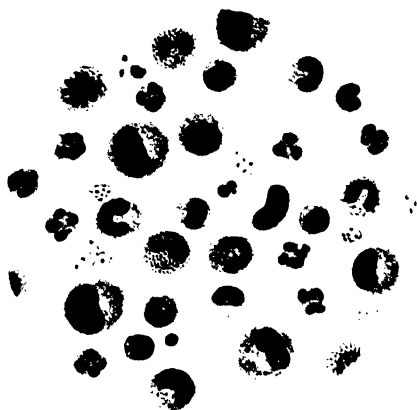
The spleen is greatly enlarged, weighing from 2 to 18 lb., and there may be enlarged splenunculi in the hilum. Its shape is preserved, but the notches are exaggerated. The surface may show fibrous plaques and adhesions to the diaphragm. In rare instances the

spleen, after being unduly movable, becomes fixed in a distant part of the abdomen, such as the pelvis or even the right iliac fossa. On section it is fairly uniformly red in colour, but often contains infarcts, and sometimes hæmorrhagic areas. The Malpighian bodies are obscured, and there is fibrosis varying according to the duration of the disease. Usually firm and tough, the organ is sometimes friable, and exceptionally rupture has occurred. Microscopically, the appearances resemble those of the bone-marrow (myeloid transformation). The pulp is crowded with myelocytes and other forms of leucocytes, and contains some giant cells of the bone-marrow. Cases of some standing present fibrosis and macrophages containing hæmosiderin.

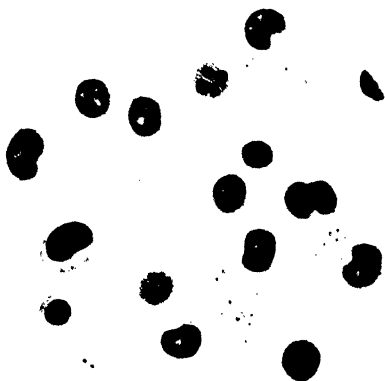
The lymphatic glands are slightly enlarged and show varying degrees of myeloid transformation.

The liver is uniformly enlarged, smooth, and pale in colour; it commonly weighs 5 or 6 lb., but may be much heavier. Section may reveal small spots due to masses of myelocytes in distended capillaries, which to the naked eye resemble miliary tubercles. The term "nodular leukæmia" has been employed to describe cases of the disease presenting tumour-like masses (Gordon Ward). In very rare instances there are tumour-like masses in the spleen and liver due to hæmorrhage, but resembling sarcoma to the unaided eye. Infiltration occurs in the kidneys and other organs, and may cause fatty change in the myocardium. In very rare cases destructive changes in the bones cause metastatic calcification in the heart.

Symptomatology.—This chronic disease may be latent for a considerable time, and its onset is so gradual that the spleen is nearly always much enlarged when the patient is first seen. The earliest symptoms may be abdominal pain, discomfort, or swelling due to the splenomegaly, or shortness of breath and weakness, or in rare instances colic from uric-acid gravel. Loss of weight, anæmia, and irregular bouts of fever, commonly to 102° F., follow. The spleen steadily increases in size, and eventually becomes enormous. Attacks of perisplenitis may give rise to pain and friction. Sudden increase in size with pain may be due to hæmorrhage into its substance. Suppuration of a splenic infarct is very rare, but I know of a case in which it gave rise to an abscess at the umbilicus and fatal purulent peritonitis. Before death or after treatment the spleen may get smaller; the liver is usually enlarged,



1



2

PLATE 16.—THE BLOOD IN (1) CHRONIC MYELOID LEUKÆMIA,
(2) ACUTE LYMPHOID LEUKÆMIA. \times 500.

smooth, and painless. Ascites is rare, and jaundice most exceptional. The stomach may be embarrassed by the large spleen, but, as a rule, flatulence and diarrhœa are rather late manifestations, though they may be due to arsenic. Hemorrhages, usually small, especially epistaxis, occur at some time or other in most cases. True hæmoptysis and hæmaturia are rare, but cerebral or gastro-intestinal hæmorrhage may be fatal. I have seen bleeding from small duodenal ulcers cause death.

In some cases there is moderate enlargement of the lymphatic glands. Corresponding to periods of excessive destruction of leucocytes there may be excess of purin bases and deposits of uric acid or gravel in the urine, but gout and symptoms ascribed to uricæmia do not occur. Late in the disease albuminuria is not uncommon, and albumosuria is occasionally reported. In rare instances albuminuria, apparently due to pressure exerted on the left renal vein by the spleen, occurs when the patient lies in bed but disappears in the erect posture. Persistent priapism may be an early complication. Leukæmic infiltration and hæmorrhages in the retina are common, and occasionally similar changes in the semicircular canals cause Menière's symptoms. Deafness may be due to these lesions in the cochlea or middle ear, or to leukæmic infiltration of the auditory nerve.

Œdema of the feet, faintness, and increasing debility are apt to occur with advancing anæmia. The heart may be displaced upwards by the large spleen, and hæmic murmurs may be heard. Purpura and leukæmic tumours due to infiltration of the skin, which may occur in lymphoid leukæmia, are most exceptional. The long bones may be tender on percussion.

The blood (PLATE 16, Fig. 1).—The total volume, as shown by the distended vessels, is probably increased. The blood coagulates badly. The leucocyte count is enormously increased, the average number per c.mm. being 400,000, and out of 71 cases 5 had counts of over a million (Cabot). The characteristic feature is the presence of myelocytes, constituting from 20 to 50 per cent. of the white cells, with an average of 35 per cent. Myeloblasts—or the non-granular precursors of the myelocytes—are present in small numbers, but shortly before death may be numerous. In cases of so-called mixed leukæmia these cells have probably been regarded as large lymphocytes; in like manner the reputed transformation of myeloid into lymphocytic leukæmia is

probably due to the occurrence of a well-marked myeloblastic change. It therefore appears that there is no necessity to recognise the existence of "mixed leukæmia," or the transformation of myeloid into lymphoid leukæmia. The bulk of the myelocytes have neutrophil granules, but some have eosinophil or basophil granules.

The polymorphonuclear neutrophils constitute about 50 per cent. of the total white count. Basophil or mast cells are increased from 0.5 up to 3 per cent. or higher; eosinophils are often increased relatively as well as absolutely. Charcot-Leyden crystals may separate from the blood, and are probably derived from the eosinophils. The lymphocytes, although relatively diminished, are absolutely increased. As the result of treatment, or spontaneously, the total leucocyte count may diminish greatly or even become normal—an aleukæmic phase—but a differential count still shows the presence of leukæmia. In rare cases the leucocyte count may fall almost to zero before death. Secondary anæmia, which may become grave, is a comparatively late event; the colour index is low, about 0.6. Nucleated red corpuscles (erythroblasts), usually normoblasts, but occasionally megaloblasts, are present. The blood-platelets are greatly increased in number. When the anæmia is very severe the blood picture may suggest a combination of leukæmia and pernicious anæmia—leukanæmia. The resistance of the erythrocytes to hypotonic solutions of salt is normal, except in the rare cases with jaundice, in which it is much diminished.

Death may be due to increasing weakness with cardiac failure and pulmonary œdema, or to some complication, such as pneumonia, cerebral or gastro-intestinal hæmorrhage, or general tuberculosis.

Complications.—Pyogenic infections, either local, such as boils or erysipelas, or streptococcic septicæmia, may occur. Local abscesses sometimes supervene after hypodermic injections of arsenic. The occasional occurrence of pneumonia is interesting in connexion with Parkes Weber's suggestion that pneumococcic inflammation is favoured by arsenic medication. Infections often, but not invariably, lead to a great fall in the leucocyte count, but without any improvement in the patient's general condition. Tuberculosis, generalized or pulmonary, is not an uncommon complication, and in rare instances a terminal generalized tuberculosis may be associated with a fall of the leucocyte

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count to normal. As leukæmic patients may fail to produce antibodies to an acute infection, there may be no agglutination in enteric fever or after antityphoid inoculation (K. M. Howell).

Prognosis.—The disease lasts from one to three years, longer in adult patients, and shorter in children, in whom it is usually fatal under a year. A leukæmic woman may bear a healthy child, but pregnancy is likely to prove fatal. Although there may be improvement and remissions, spontaneous or due to treatment, the disease must be considered as universally fatal.

The number of leucocytes is not a trustworthy guide to the duration of the disease, but the appearance of large numbers of myeloblasts in the blood is a sign that the end is near. When an acute infection occurs, the outlook is bad.

Diagnosis.—From other conditions with considerable splenomegaly the diagnosis is easily made by a complete blood-count. In the rare cases in which the blood becomes normal a diagnosis cannot be made during this phase.

Treatment.—Arsenic in large and continuous doses is often followed by improvement or remission, but subsequently fails to have any effect. In cases intolerant to arsenic by the mouth, hypodermic injections may be well borne, but the intravenous injection of salvarsan preparations does not appear to have been successful.

X-ray treatment to the long bones and spleen, like arsenic, is followed by remissions and subsequent failure. Radium exposures in massive doses appear to give better results than X-rays. Treatment with arsenic may with advantage be combined. Usually, when the total leucocyte count falls, the differential count remains characteristic of myeloid leukæmia.

Benzol was employed on the basis of Selling's observations that it produces leucopenia which persists after its administration ceases. At first the leucocyte count is increased; but subsequently the number of leucocytes diminishes both from their destruction in the blood and from inhibition of the leucoblastic tissues, while the red blood-count and hæmoglobin increase. The effect of benzol appears to be less rapid than that of X-rays. It should be mixed with olive oil, and 2 to 3 c.c. given daily in gelatin capsules after meals and with milk, so as to avoid gastric irritation. It acts equally well in myeloid and lymphoid leukæmia, but it

should be avoided in acute cases with much fever and also in those with much leukæmic retinitis; and, as its effect continues after its administration is stopped, a diminution in the leucocyte count is an indication for the cessation of treatment. Most cases relapse after the treatment is stopped. In a comparatively acute case an apparent cure followed a course of naphthalene tetrachloride 8 gr. (J. H. Drysdale).

Splenectomy in the past has been almost always fatal from hæmorrhage; up to 1915 there were 51 splenectomies with 8 temporary survivals, or an operative mortality of 81·9 per cent. But in 1916 (Griffin) advocated preliminary radium treatment of the spleen, with subsequent splenectomy; at the Mayo clinic, out of 20 myeloid leukæmic patients submitted to splenectomy, 18 having previously had the radium treatment, there was 1 operative death, and 10 were in good health a year later.

ACUTE LYMPHOID LEUKÆMIA (*syn.* Acute Lymphatic or Lymphocytic Leukæmia; Acute Lymphæmia)

An acute febrile disease characterized by great excess of cells of the lymphocyte class in the blood, liability to hæmorrhages, and a rapidly fatal termination.

Etiology.—It is much commoner under the age of 30 than after; of 100 cases, 20 per cent. occurred in the first decade, 22·5 in the second, and 26·1 in the third, or 68·6 per cent. under 31 years. Males are more often attacked; out of 100 cases, 70 were males (Boudet). Reported cases of congenital leukæmia are open to doubt. It may be difficult to decide if a given case should be regarded as acute, or as chronic with an acute termination. The limit for an acute case has been considered to be nine weeks, but in some instances the onset of acute symptoms has been preceded by enlargement of the glands or spleen.

Morbid anatomy.—The bone-marrow is red, as in fatal life, or soft and palish-yellow (pyoid), but its condition varies in different parts, and the change may be confined to a few bones. Exceptionally there is rarefaction of the bones. Microscopically the marrow is infiltrated with primitive cells. The lymphatic glands are soft, without periadenitis, and on section white with scattered hæmorrhages. Microscopically the dense infiltration with lymphocytes obscures the normal structure. The spleen is enlarged, soft or diffident, and shows lymphocytic infiltration. The thymus

LEUKÆMIA

is often enlarged; this has been interpreted in various ways—as a secondary infiltration, as a primary sarcoma with secondary leukæmia allied to Sternberg's leucosarcoma, or as showing a definite relation between the status thymo-lymphaticus and acute lymphocytic leukæmia. The liver is usually enlarged, but to a moderate degree only. There is periportal infiltration with lymphocytes. The kidneys show lymphocytic infiltration and are sometimes greatly increased in size. Hemorrhages, multiple or single, are occasionally seen in the brain.

Symptomatology.—The onset is usually gradual, with lassitude, fever, and anæmia; less often symptoms come on acutely with hemorrhages, sloughing tonsillitis, or stomatitis, so that the condition may at first be regarded as purpura hæmorrhagica, scurvy, or gangrenous or ulcerative stomatitis and gingivitis, enteric or other fever. In such cases glandular enlargement may be absent. Any considerable splenomegaly shows that the disease, though now acute, has really existed for some time. The aspect of the disease varies with the prominence of grave anæmia, hemorrhages, or stomatitis. The first symptoms may be somewhat misleading, namely, hæmaturia, metrorrhagia, pain in the limbs or back. The anæmia is progressive, cardiac murmurs appear, there are fever which may reach 105° F., sweating, increasing weakness, and loss of weight. As time goes on, some enlargement of the glands and spleen is usually detected, and Mikulicz's disease may be simulated. The liver may enlarge, in very rare cases without splenomegaly. The urine commonly contains albumin, sometimes blood and casts, and the uric-acid content may be increased. Hemorrhages are frequent in all parts, including the retina and the brain, and may prove fatal. In exceptional cases there may be experienced severe pain in the bones.

The blood (PLATE 16, Fig. 2).—The erythrocytes diminish in numbers, even to one million or less. Poikilocytes, normoblasts, and megakaryoblasts are usually present. In the early stages the colour index is low, but as the anæmia becomes grave it tends to rise. The platelets are diminished in number. The leucocyte count is not so high as in myeloid leukæmia, the average being between 100,000 and 200,000; occasionally it is very high, and has even been known to exceed that of the reds. On the other hand, there may be but slight increase in the total white count, so that the disease is

only recognized by the differential count. The predominating white cell is a non-granular mononuclear cell, usually spoken of as a lymphocyte, but atypical and embryonic, thus resembling the lymphoblasts; it constitutes from 90 to 99 per cent. of the total white count. It is generally agreed that a large cell with frayed edges is characteristic of acute leukæmia, but this is not universally true, for in some cases of acute leukæmia the predominating cell is a small lymphocyte, as in most cases of chronic lymphoid leukæmia.

Diagnosis depends on the blood examination, and in its absence cases without glandular or splenic enlargement may be regarded as gangrenous or ulcerative stomatitis or gingivitis, purpura fulminans, scurvy, enteric or other fevers. Even with a blood-count there may be difficulty if the high lymphocytosis of whooping-cough is not borne in mind. In addition, some cases of wound infection, boils, and widespread streptococcal adenitis of tonsillar origin may be accompanied by a lymphocytosis suggesting lymphoid leukæmia (Cabot, Marchand); the distinction rests on the recognition of an infective origin for the adenitis, the lesser degree of lymphæmia, and the course of the disease. Lymphosarcoma, Sternberg's leucosarcoma (in which condition there is a blood picture of lymphoid leukæmia with an invasive lymphoid tumour), and lymphoid leukæmia have been thought to be different manifestations of the same disease (L. T. Webster).

Prognosis.—The disease runs an extremely rapid course, and may prove fatal in as short a time as a week from the onset of symptoms. Out of 54 cases in children, 8 survived for more than two months (Thursfield). The outlook must be regarded as absolutely fatal. Two cases of cure of what appeared indubitably to be acute lymphoid leukæmia are known to me, but in such exceptions the exclusion of the extreme lymphocytosis of infection is a difficult problem.

ACUTE MYELOID LEUKÆMIA

This form of leukæmia was formerly considered to be extremely rare, because the myeloblasts or the non-granular precursors of the myelocytes were regarded as large lymphocytes. Since this fallacy has been avoided, the comparative frequency of the disease is becoming recognized; thus, among 59 consecutive cases of leukæmia there were 16 of acute and 29 of chronic myeloid leukæmia, and 8 of acute and 6 of chronic lymphoid leukæmia

LEUKÆMIA

(Panton, Tidy, and Pearson). Some exceptional instances of miliary tuberculosis show acute myelæmia (Marshall). The clinical features and the prognosis of acute myeloid are the same as those of acute lymphoid leukæmia.

It has been suggested that the oxydase reaction, namely, the detection of oxydase ferment granules, which are only found in cells from the bone-marrow, may be a guide to the form of acute leukæmia, but this criterion is not universally accepted.

Treatment of acute leukæmia.—This is most disappointing. X-rays are said to do harm; transfusion of blood is followed by considerable but quite transient improvement.

CHLOROMA (*syn.* Chloro-leukæmia)

An atypical leukæmia, nearly always acute and of the lymphoid form, though a few examples of myeloid or myeloblastic chloroma are on record. The characteristic feature is the presence of greenish tumours in connexion with bone, particularly about the orbits and temporal fossæ, which produce proptosis, and in the vertebrae, ribs, and viscera. Like acute leukæmia, it especially attacks males and young persons. Other manifestations are weakness, anæmia, hæmorrhages, enlargement of the liver and spleen, deafness, blindness, and pain, especially in the limbs. The cause of the green colour, which usually fades rapidly on exposure and need not be present in all the tumours, is unknown; it has been variously explained as due to a lipid, to altered blood pigment, to granules in the cells such as eosinophil granules, or to degeneration products. This disease is of interest in connexion with the relation of ordinary leukæmia to sarcoma, and it might be suggested that there are transitions from ordinary sarcoma, through myeloma, Sternberg's leucosarcoma, and chloroma, to leukæmia. Proptosis and orbital hæmorrhages may occur in infantile scurvy and as the result of metastases in malignant hypernephromas, but a blood-count will settle the diagnosis. Chloroma runs a rapid course in a few (usually two to four) months, though a duration of eighteen months has been recorded. In a case of acute leukæmia the presence of proptosis or of tumours in the orbits and temporal fossæ justifies a diagnosis of chloroma; but, as non-chloromatous tumours in these situations sometimes occur in acute leukæmia, a dogmatic diagnosis cannot be so well in during life.

CHRONIC LYMPHOID LEUKÆMIA

An afebrile disease of chronic course with a great excess of lymphocytes in the blood.

Etiology.—It occurs in adult life, beginning after the age of 30, and is more frequent in males than in females. In early life, however, cases occur in which the lymphatic glands have been enlarged for months before the onset of acute leukæmia. It is often associated with a focal infection, commonly in the mouth. It is usually stated to be the least frequent form of leukæmia, but Vogel regards it as less rare than acute lymphoid leukæmia. It was first thought to arise in the lymphatic glands, but, as the bone-marrow is now believed to be responsible, the name lymphoid or lymphocytic leukæmia is more suitable than lymphatic leukæmia.

Morbid anatomy.—The bone-marrow has either a pale-red or a greyish-red colour, and microscopically it shows intense lymphocytic infiltration. The lymphatic glands are soft, separate, and not matted together as in lymphadenoma, and are succulent and white or pink on section. Sometimes the lymphatic tissue of the alimentary canal is widely and considerably affected, and the vermiform appendix may be very much thickened. Microscopically a diffuse lymphocytic infiltration obscures the structure of the glands. The spleen is increased in size. The follicles are enlarged and the pulp is overcrowded with lymphocytes. The liver is uniformly enlarged, somewhat pale, and often shows spots like tubercles due to masses of lymphocytes. Occasionally there is widespread amyloid change in the viscera. I have seen hypoplasia of the aorta, but the association is not constant.

Symptomatology.—The onset is very gradual, and enlarged glands without constitutional symptoms may have existed for a long time before the disease is recognized. In other cases abdominal discomfort or distension due to the enlarged spleen leads to the discovery of the condition. The general aspect of the disease is much the same as that of chronic myeloid leukæmia, but usually there is more evidence of glandular enlargement and its course is more chronic. The splenomegaly, though less than in the chronic myeloid form, may be considerable. The enlarged lymphatic glands are discrete, often soft, and very seldom reach a large size or cause pressure symptoms. They are usually first noticed in the neck, then in the axilla, and less often all over the body. In rare instances there is no palpable enlarge-

LEUKÆMIA

ment of lymphatic glands. The liver is not so often enlarged as in the myeloid form. As time goes on there is some loss of weight, and late in the disease anæmia appears. Fever, too, may appear at a later stage; hæmorrhages and purpura are rare unless the condition becomes acute. Other cutaneous changes are rare. There may be definite tumours which in some instances resemble mycosis fungoides, or flat nodules scattered over the body, or diffuse œdema-like infiltration. On the face confluent masses may produce a leonine appearance. The tumours may change or disappear. Pruritus in association with prurigo-like papules, pityriasis rubra, or general exfoliative dermatitis has been described, as in lymphadenoma, in which it is certainly more often seen.

The blood.—The total white count is usually less than in chronic myeloid, but generally higher than in acute lymphocytic leukæmia. The predominating cell is a lymphocyte, which forms from 80 to 90 per cent. of all the white cells. It is often stated that the characteristic cell is a small lymphocyte, whereas in the acute cases the predominating cell is a large lymphocyte; but this statement is much too dogmatic, for large lymphocytes may be the predominating cell in chronic cases; it has recently been urged that acute and chronic lymphoid leukæmia cannot be differentiated solely by examination of a blood-film.

Until the late stages, when a secondary anæmia supervenes, the red blood-cells and hæmoglobin are fairly normal. Nucleated erythrocytes are not present.

Prognosis.—The duration of life is longer than in the other forms of leukæmia; it may be five years or more, and Osler mentions a duration of at least ten years. On the other hand, it is less influenced by treatment than the chronic myeloid form. An apparently complete remission has been reported (Gulland and Goodall).

The **diagnosis** depends on a blood-count, which distinguishes this condition from lymphadenoma and other forms of lymphatic-gland enlargement. From examination of the blood alone, confusion might possibly arise with lymphocytosis due to other causes, such as whooping-cough, or, in rare instances, to streptococic infection.

Treatment is on the same lines as in chronic myeloid leukæmia. Some definite improvement has followed intravenous transfusion of human blood (Otterberg and Libman).

H. D. ROLLESTON.

LICHEN PLANUS

LEUKANÆMIA.—This word has been applied to cases with a blood picture combining the features of leukæmia and pernicious anæmia. Critical examination of the cases shows that it is a phase either in the course of leukæmia or, more rarely, of pernicious anæmia, and not a distinct disease. In the late stages of leukæmia there is commonly anæmia more or less of the pernicious type, and patients with pernicious anæmia may show a terminal lymphocytosis. This apparent combination of leukæmia and pernicious anæmia is especially likely to occur in children.

H. D. ROLLESTON.

LICE (see PEDICULOSIS).

LICHEN OF THE TONGUE (see STOMATITIS AND GLOSSITIS).

LICHEN PLANUS.—A disease characterized by the appearance on the skin of groups of small infiltrated papules, usually flat-topped and of angular outline.

Etiology.—The disease has long been known to be associated with nervous disturbance of obscure nature, and has been observed to appear suddenly after severe mental shock, and, in the more chronic form, after prolonged worry. Radcliffe Crocker was of opinion that sudden chills during perspiration determined the onset of the eruption, and in two of my cases there was a very clear history of this etiology. The disease is rare in young children. Most cases undoubtedly occur in the period extending from early adult life to middle age, but it is not uncommon in old age. Women are affected rather more frequently than men, and the disease is distinctly more common among brainworkers than in the labouring classes.

Pathology.—The disease appears to be an inflammation of the superficial part of the true skin, and is generally connected with sweat-ducts or hair-follicles. The anatomy of the excised papule shows that the disease affects both the papillary layer of the corium and the epidermis. In the former there is found a collection of oval endothelium-like cells which here and there may be seen to be derived by budding from the capillary endothelium. Between the summits of the papillæ and the basal layer of the epidermis there is generally a small cavity, sometimes called "the lacuna," which is filled with serum and perhaps a small amount of blood. In certain cases this space becomes so much enlarged as to give the clinical appearance of a bulla. The changes in the epidermis

LICHEN PLANUS

are confined to a slight increase in the thickness of the mucous layer, an irregularity in the granular layer, and a hypertrophy of the horny layer which is very variable, being in the ordinary plane papule only slight, but reaching a much greater degree in the follicular papule and forming pumice-stone-like masses in the verrucose form of lichen planus.

Symptomatology. (1) **The eruption.** (a) *On the skin.*—The characteristic element of the eruption is a small tough papule, varying in size from that of an ordinary pin's head to $\frac{1}{8}$ in. in diameter. Usually it is angular in shape, more rarely almost round.

The papule is raised slightly above the normal surface, has a flat top and shining surface, and offers a sense of resistance to the finger (infiltration). In colour it is, when recent, of a lively bluish red or "lilac" shade, but older and fading papules are often purple from added pigmentation and may be quite brown. The red colour of the papule is not quite even, but there are minute wavy lines of milky white coursing over its surface. These lines, known as "Wickham's striae," are produced by the irregular formation of the granular layer mentioned above. The papules are grouped in short curved lines, a linear arrangement which may be greatly exaggerated as described below. Not infrequently small rings are formed by the grouping of papules, and much less commonly rings are produced by the actual peripheral enlargement of single papules followed by central involution. Ring-formation is much commoner on the penis than elsewhere. With the involution of the lesions, a definite atrophy of the true skin sometimes occurs. In some cases scaling, always secondary in nature, is somewhat pronounced, so that a superficial resemblance to psoriasis may be present. Lastly, formation of bullæ may take place. In this case the whole of the epidermis is raised up from the papillary body so as to form a thick-walled blister, the contents of which are often bloodstained, while the roof shows the markings of the original papules. This appears in some cases to be due to the administration of arsenic.

The second variety of papule is that which occurs around the hair-follicles. In this case the papules are very small, round in outline, and either carry a minute horn or show a central depression where the horny mass has dropped out. Such papules may or may not be grouped, but at any rate they seldom show the striking patterns seen with the plane

variety. They may run together to form masses of various sizes, and by enormous hypertrophy both of the corium and epidermis may produce warty masses known as *lichen planus hypertrophicus*.

Both forms of papule may be present together (*lichen plano-pilaris*), but it is not infrequent for the follicular papules to occur alone for a time, either before or after the plane papules, in which case the diagnosis becomes difficult.

The eruption may attack the whole trunk and extremities, including the face, but there are certain sites of predilection. These are the fronts of the wrists and the inner sides of the thighs near the knees, the forearms and shins, and the waist line and glans penis. The face usually escapes, and the eruption is said to be very rare on the hairy scalp, but my experience leads me to believe that it is commoner here than is generally stated. The sites of predilection on the scalp are the parts immediately above the ears and that below the occipital protuberance. In these situations the eruption forms discoid, scaly patches with some infiltration, and is sometimes diagnosed as scaly eczema.

There is also the curious form known as *linear lichen planus*, in which long streaks of the eruption occur. These often suggest a nerve distribution, but as a matter of fact in most cases they do not accurately correspond with the anatomical distribution of any known nerve. In a striking case of mine the eruption consisted of two streaks only. One began on the right side of the pubic region, passed along the right inguino-scrotal fold, appeared below the gluteus maximus, ran down the middle of the thigh and leg, and ended just behind the inner malleolus. The other began immediately above the right anterior superior iliac spine and followed closely the course of the sartorius muscle to end on the inner side of the thigh above the knee.

Any slight trauma such as pressure or scratching will determine the appearance of the eruption in lichen planus, but streaks thus produced are not to be confused with those in the essentially linear cases.

(b) *On mucous membranes.*—Lichen planus is one of the rather limited number of diseases of the skin which also affect the mucous membranes. The commonest part to be affected is the buccal mucous membrane, but it is frequent on the tongue and not uncommon on the anus, while the vaginal mucous membrane has been

LICHEN PLANUS

also affected. In these situations the eruption presents itself as a number of small pearly white dots and lines arranged in a fine network. Scraping with a blunt instrument does not remove any of the white marks, and the finger hardly detects any sense of resistance, a slight harshness being the only palpable sign. The patient is generally unaware of the eruption in the mouth, but occasionally there is a feeling of soreness, especially when the tongue is affected.

(2) **Subjective and general symptoms.**—In some of the most widespread eruptions other symptoms are absent. As a rule, however, itching is severe, and may be so distressing as to keep the patient entirely from sleeping. Not uncommonly also there are general symptoms of excitability of the nervous system, the patients becoming excessively emotional and bursting into tears while relating their symptoms.

Diagnosis.—Attentive and close study of the eruption will in most cases lead to the correct opinion, but the following are points to which especial attention may be given: In *papular eczema*, *lichen urticatus*, and *sudamina* confusion is only possible when the papules of these diseases are undergoing involution and flattening down. They are then round in shape, the shiny surface is seen on the slightest violence to become moist, there are no white striae present, and the consistency of the papule is less tough than in lichen planus. *Psoriasis* may be distinguished by the fact that the scaling is primary, and that on removal of the scales there is left a bright-red surface which bleeds in minute points on being lightly scratched. The *lichenoid syphilide* is apt to be accompanied by other signs of secondary syphilis, the papules are round, are grouped in discoid patches rather than in lines, and are situated most thickly on the shoulders. The *tuberculide* shows a similar grouping to that of the syphilide, and more staining remains in the papules when the hyperæmia is pressed out. Of course, in these eruptions a Wassermann reaction or a tuberculin test may be carried out if desired. *Pityriasis rubra pilaris* does not often give rise to difficulty, though at one time there was considerable confusion. The chief points to be relied on are the scaling and the hyperkeratosis of the palms, the blackened hyperkeratosis of the follicles of the fingers, the stalicite masses of scales on the knees and elbows, and the involvement of scalp and face. From *lichen spinulosus* the disease can only be

diagnosed by a careful search for the plane papule and possibly by the history; it is doubtful at present whether many of the cases called lichen spinulosus have not been in reality follicular lichen.

Prognosis.—Lichen planus in itself is not dangerous to life, but in acute cases the suffering may be very severe and the mind may be almost unhinged. The duration of a generalized attack may be given as from two to six months or longer. The hypertrophic patches show practically no tendency to get well spontaneously and may be seen years after the subsidence of the attack with which they were associated. In very rare cases the disease may run on into dermatitis exfoliativa, in which event the result may be fatal.

Treatment.—Two drugs stand out pre-eminently in the internal treatment of lichen planus, namely, arsenic and mercury. In very acute cases with great irritation arsenic is, as a rule, contraindicated, as it increases the irritation and may actually bring out more of the eruption. In less acute cases and in those in which inflammatory symptoms are not pronounced it is useful in subduing the eruption. It must be used in full doses, and may be begun in 5-min. doses of the liquor and quickly increased until 10 or 12 min. three times a day are taken, care being taken, of course, to avoid upsetting the patient. In any case the drug should not be continued too long, a period of two months being generally sufficient. Mercury may be used in the more inflammatory attacks, and the perchloride and biniodide have appeared to me to be the most useful forms. Large doses are not required, and $\frac{1}{16}$ gr. of the perchloride, with or without 10 gr. of potassium iodide, three times a day is usually sufficient.

When the irritation is intense it is all-important to procure sleep. For this purpose antipyrin or phenacetin may be tried first, and if these fail preparations of opium may be used.

Salicin in large doses was recommended strongly in the more acute cases by Radcliffe Crocker, but it has proved generally disappointing in my hands.

Recently a new form of treatment has been introduced, namely, the withdrawal of a small amount (5-7 c.c.) of cerebro-spinal fluid by means of lumbar puncture. Excellent results have been published from the use of this method, and in the cases in which I have used it the effect was almost miraculous, all the symptoms in very severe cases subsiding

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in a few days after the withdrawal. One would not, of course, advise this method as a routine treatment, but in very severe or obstinate cases it would seem to be quite justifiable.

Local treatment.—This is not so important in most cases as the general treatment. For the ordinary type of plane eruption weak tar lotions seem to be the most efficacious, such as 1 dr. of liquor picis carbonis in 8 oz. of calamine or lead lotion. Unna recommends an ointment containing from 2-5 gr. of perchloride of mercury and 10-20 min. of liquid carbolic acid in the ounce of zinc ointment, but I have not had much success with it.

In the follicular type with spines a 5 per-cent. salicylic-acid ointment well rubbed in is usually rapidly efficacious.

For the hypertrophic patches, only very energetic measures are likely to be successful. Strong (20 per cent.) salicylic-acid plaster may be applied to each of the patches, or 10-per-cent. pyrogallol ointment may be rubbed in; but perhaps the best method is to give repeated pastille doses of X-rays at three-weekly intervals. Under this treatment involution generally takes place rather rapidly, and the intolerable itching ceases soon after the first dose.

ARTHUR WHITFIELD.

LICHEN SCROFULOSUS (*see* TUBERCULIDES).

LICHENIFICATION (*see* PRURIGO).

LIFE INSURANCE.—In this article the routine method of conducting an examination and making a report by a medical referee will be described, and those points emphasized which experience has shown to be of chief importance. The medical referee must have all his wits about him in this work, because he has to deal with presumably healthy men, and he seldom meets with any very gross lesion. In the vast majority of cases the signs are very slight and have to be looked for carefully.

General inspection.—A good light is essential. All cases should be examined in daylight, with the examiner's back to the window and the subject facing it.

Family history and heredity should be first considered. Family tendencies to long or short life are of great importance in estimating expectation of the lives of proposers for insurance. In addition to a tendency to longevity, members of such families bear acute illnesses better than those who have no such family record, and other severe complaints are also

better borne. On the other hand, there are short-lived families, of which several members die before 60; in these chronic nephritis, high blood-pressure, cardio-vascular sclerosis, and apoplexy are not uncommon causes of early family deaths. The early death of both parents should always be considered, even if the diseases were not hereditary, for it suggests an inability to withstand ordinary ailments. Of still greater significance is the early death of both parents from similar complaints, of whatever nature.

A **tuberculous** family history is one of the most common complications of insurance, and may relate to one or more members of the family. Deaths from "bronchitis," "general decline," "childbirth," should be carefully investigated. With a family history of tuberculosis the insurance risk of the development of tuberculosis is considerable; the previous medical history of the proposer, and his own physical condition, his nutrition, and his habits, must be noted with great care. All these must be unexceptionable for the issue of a policy to be thought of on any terms.

As a general rule, it is unsafe to insure anyone for a tuberculous risk *before the age of 30*, however well nourished the proposer, however well-shaped and movable the chest, however clean his medical history, and however good his habits.

The risks of an hereditary tendency to **cancer** are not so great as those of tuberculosis. As a rule, the death of one or other parent from this disease will not adversely affect a proposal. If both died at a good age, there is no need to weight the premium. If both died at an early age, some addition to the premium might be called for; and if one or more brothers and sisters died from cancer at an age above that of the proposer, this would be the more necessary.

Diabetes mellitus in one or both parents must be noted. I have found sugar in the urine of several proposers with this family history.

High blood-pressure, from whatever cause, also runs in families.

Gout is not often found now in the family history of commercial classes.

The **medical history** of the proposer for insurance is important. Chief among previous medical ailments are diseases of the lungs and pleura, rheumatic fever, and syphilis. Of pulmonary diseases, **pleurisy** is the most important, as it is so often tuberculous in origin. If it dated from several years pre-

viously and has left no traces, it may be ignored ; but if it occurred within recent months or has left its mark on the lungs or the pleura, the risks of insuring the life are generally too great for the issue of a policy.

Rheumatic fever.—One attack of this disease, occurring years before, which has not affected the heart, may be ignored, but not so recurring attacks. If there is any valvular lesion the risk from a subsequent attack is grave.

A previous history of **malaria** or **influenza** is not uncommon in aortic valvular heart disease.

A history of **gonorrhœa** is common. If the cure is complete it need not affect the insurance.

Syphilis is less common, and is certainly not acknowledged in 5 per cent. of proposals. When there is a history of it, or any suggestion of it is found during the examination, e.g. leukoplakia, abnormal pupils, etc., a Wassermann test must be done, and, if positive, insurance refused.

Alcohol.—If there is any smell of alcohol in an applicant for insurance, the examiner must proceed cautiously. One glass of beer or sherry may make the breath smell, but will not cause a quick, full, alcoholic pulse. When such conditions are met with after lunch, the probability is that too much alcohol is habitually taken. A history of alcohol taken in business hours or between meals is suspicious. When the proposer takes nothing until after seven o'clock in the evening, the amount then taken should be ascertained. If drink is taken at a club or public-house and, judging from physical signs present, the amount taken is probably understated, I advise refusal of a policy. Generally, such persons have a badly congested smoker's fauces as well.

Physical examination.—The pulse should be felt at the beginning of the examination, for it may at once indicate the value of the proposal. The rate, rhythm, volume, condition of the arterial wall, and the tension must be noted. The tension is most important. A sphygmomanometer is not necessary for anyone who has had fair clinical experience, but some companies require its use always, or at any rate in men over 50. Any increase of tension will probably be accompanied by a history of nocturnal polyuria, and possibly also by arteriosclerotic changes in the vessel-wall. In these cases albumin should be sought for with the greatest care, the acidulation and boiling test

being used. The nitric-acid test in the cold is not delicate enough. Albumin is frequently absent in chronic high tension, the cause of this being toxic and not necessarily renal.

Changes in the vessel-wall and increase in the tension of the pulse are most likely to be met with after the age of 50, but may be encountered before. People may live to old age with great increase (200 mm.) in tension and a trace of albumin in the urine, but it is impossible to tell which of them will do this, and insurance companies are very unwilling to issue policies for them. If there are any other unfavourable indications, e.g. a family history of early death from apoplexy, or if there is a suspicion of alcoholic or dietetic excess, refusal would be certain.

Height and weight.—Family history is an important factor in judging the effect of height and weight on prospects of life. Undue heaviness or lightness often runs in families. The age and weight of parents and grandparents are of importance here, and other conditions also must be taken into consideration, such as a family history of tendency to disease, especially phthisis, the shape and capacity for expansion of the proposer's chest, and his habits and constitution generally. Progressive increase or loss of weight must also carefully be inquired into. Marked underweight for height (25-45 lb.), considered alone, is more risky in people below 30 than similar overweight, because their powers of assimilating food are probably below the average ; while the reverse holds good for adults over 50. People of nervous temperament, worriers, often quite good lives, are frequently well below weight. The size of the bones and the nature of the general build are important, some families being much more lightly built than others.

Stout people are often big eaters and drinkers, and do not take much exercise. They stand acute illnesses badly. They are bad lives, especially when found amongst publicans, cattle dealers, butchers, commercial travellers, or any occupation which leads to excess in consumption of alcohol and to a sedentary life. Diabetes often supervenes. Great excess of weight (50-80 lb.) is bad at all ages. (Edema of the shins is often present in such people. Women carry stoutness better than men, for they are more careful in their habits and less subject to risks in everyday life.

The best height is about 5 ft. 10 in. Very tall men, on the average, do not live so long as men of average height ; but height itself

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rarely enters into the question of the eligibility of a life for insurance. Midgets and giants are both rare in insurance proposals.

The **cardio-vascular system** should always be examined in a systematic and thorough way. The position and nature of the impulse or apex beat gives valuable information. If the impulse is not felt at first, I endeavour to make it more palpable by altering the position of the chest and asking for forced expiration. In high tension the heart may be twice or thrice the normal size, and yet supposed to be healthy.

The most important factor in deciding on the value of an impaired heart for life insurance is the condition of its muscle, and its power to carry on the circulation properly and to respond to the stress and strain of illness. There must be no sign of cardiac-muscle failure. Abnormalities in the sounds of the heart—accentuation, reduplication, weakening, or arrhythmia—absence of diastolic rest of the ventricle, or tic-tac rhythm and murmurs, must carefully be sought for.

Accentuation of the heart-sounds is not uncommon. Its significance depends upon whether there are any other indications of cardio-vascular abnormality. A loud first sound in a nervous, over-acting heart with no other abnormalities is of no pathological significance. It is important if coupled with other evidence of disease, such as cardiac enlargement, high-tension pulse, arterio-sclerosis, or aneurysm. Such cases are uninsurable.

Nervous or insurance heart.—In a regular though small percentage of people a visit for medical examination by a doctor will increase the heart-rate by from ten to sixty or more beats per minute. This very quick action of the heart constitutes the typical insurance or apprehensive heart. Many hearts will slow down entirely to normal within five minutes. Some of those which fail to slow down completely will do so partially, beating at about 80 or so for ten seconds, and then perhaps running off to 120 or more again. Such a heart may slow down and beat regularly at near the normal rate after ten to twenty minutes in the waiting-room. A small proportion may only slow down on a second visit, when the pulse-rate may be found to be normal. Finally, there remain cases that will be almost as bad on a subsequent visit as on the first, and in these there is generally a history of nervousness in the family. As a rule, they are fair risks at ordinary rates, if the proposer is otherwise thoroughly sound, of good habits, plays games,

and has no evidence of any cardiac weakness or of Graves's disease.

A quick heart with organic disease will maintain its rapidity throughout the first and subsequent interviews, and there will probably be other signs of cardiac-muscle failure. Such a case is unacceptable for insurance.

In these cases of nervous rapidity the heart's impulse is forcible, and extends nearer the nipple line than it will when it has slowed down to its proper rate. The sounds are short and abrupt, but otherwise normal and properly spaced, and there is no breathlessness, and generally no sensation of quick heart's action.

Weakening of the sounds may mean nothing, unless there are other signs of disease, as, for instance, obesity. The same may be said of their reduplication, but when this change occurs in association with signs of valvular or muscle disease or a galloping action, it becomes serious.

Cardiac rhythm.—The heart is frequently found to "drop a beat," even with no signs of valvular or muscular disease, or history of excessive smoking, as is so often supposed to be the case. This peculiarity in rhythm may be present for years, and may be a family characteristic; it is then of no importance. When, however, it occurs in a heart in which there is involvement of valve and muscle, it adds to the seriousness of the outlook.

Murmurs.—Those arising at the aortic valve are the most serious because they connote abnormal strain on the heart-muscle, and generally also enlargement of the heart. The aortic diastolic murmur should be sought for with the greatest care; though the most important, it is sometimes the most difficult of all murmurs to hear. It may be heard at the lower part of the sternum at its junction with the fourth costal cartilage, and nowhere else. Occasionally a systolic aortic murmur is heard, but no diastolic; yet whenever there is aortic obstruction there is almost always some incompetence of the valve. The presence of either form of aortic murmur almost invariably leads to refusal of a proposal. It is conceivable that there might be a slight lesion of the cusp of the valve of old rheumatic origin which creates a murmur but offers no obstruction to the passage of blood through the valve and leads to no hypertrophy of moment. With an otherwise unexceptionable history and condition a policy might be issued, with or without some addition to the premium. The possibility of a previous history of syphilis in aortic cases must be remembered.

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Aortic disease must be suspected whenever a heart is enlarged and beats regularly and moderately slowly and there is no increased arterial tension.

The harmless posture murmur which arises from pressure on the large arteries at the base of the heart or root of the neck is most deceptive and easily mistaken for an aortic murmur.

Mitral disease, with slight structural change in the valve curtains and no affection of the heart-muscle, is the least serious of heart lesions. A mitral regurgitant murmur, with heart of normal size, or but slightly enlarged by perfect compensatory hypertrophy, due to an endocarditis which began and ended many years previously, and which has caused no discomfort of any kind, is not uncommonly met with in healthy adults. There is some distortion of the valve curtains, but no marked obstruction to the orifice. If the history is otherwise unexceptionable, such a case has a value for life insurance purposes, possibly at ordinary rates of premium. I should, however, refuse or weight heavily a young person with undoubted mitral stenosis with crescendo murmur and accentuated first sound, with or without a diastolic murmur. The condition which causes the stenosis is of slowly but continuously progressive nature, and interferes with the circulation, especially that of the lungs. Intercurrent illnesses are borne badly, and these subjects do not live their normal expectation of life. I would not issue policies to run after such people reach 50.

Tricuspid murmurs are very uncommon in people supposed to be healthy. They would contraindicate a favourable report, in view of their effects on the circulation. *Pulmonary valve murmurs* are also uncommon. If due to structural changes in the valve they would indicate refusal. If met with in anæmic people, and probably due to temporary causes, they would indicate a postponement of the decision pending treatment for the primary anæmic condition.

Respiratory organs.—In examining the respiratory organs, the bare chest must be inspected, especially the subclavicular regions. The respiratory capacity—the difference between deep inspiration and expiration—must be recorded. Any of the signs which point to changes in the lungs must be looked for, such as difference in the development or movement between the two sides of the chest, flattening in any region, diminished vocal fremitus, etc.

The apices of both lungs and the subclavicular areas must be percussed carefully, but it is often difficult, and sometimes impossible, to make out the signs of very early phthisis in this way. The bases of the lungs should also be examined, especially when there is a suspicion of bronchitis, emphysema, asthma, or other respiratory disorder. The observer will be all the more acute in his search for abnormal sounds when there are other warning indications present in the family or personal history.

The respiratory capacity of the chest is taken by measuring around the nipple line the difference between full inspiration and deep expiration. In the average man this is about 2½ in.; in athletes and army-trained men it may be as much as 5 in. In abnormal conditions such as adherent pleura or pulmonary fibrosis the movement may be less than an inch. In stout people otherwise normal the movements are often slight, being sometimes even less than an inch.

Some perfectly healthy people of average weight are unable to take deep inspirations, and register about only half an inch of respiratory capacity. I doubt whether shallow breathing has any appreciable effect on the duration of life in otherwise unexceptionable lives. It becomes important when any sign of chest disease is present or when the family history in this respect is unsatisfactory.

Pleurisy.—With a previous history of pleurisy, great care must be exercised in examining the lungs. It should definitely be ascertained whether it was really pleurisy or pleurodynia that occurred. Acute pleurisy at the best takes longer than "two or three days in bed and a week off work altogether." After pleurisy, especially with fluid in the chest, it is an equal chance that phthisis will develop later in life. There may be no abiding signs in the lungs or pleura, and if the family history is good and the attack has occurred some years previously, a policy may be issued at ordinary rates. On the other hand, if definite changes are left—e.g. thickened and adherent pleuræ and their sequelæ—the value of the proposal for insurance must be prejudiced accordingly.

Pneumonia.—After two or more attacks, if the last occurred within recent years and the proposer is a young adult (below 35), I should be inclined to weight the proposal.

Bronchitis.—Candidates with a liability to bronchitis in the winter occasionally come for examination. If the bronchitis does not confine them to bed much, if it clears up completely

for most of the year, and if the family history is good, they have some insurable value. If adventitious signs, even only a few râles or rhonchi, are present at the time of examination, especially in the summer, I should refuse a policy. The family history is important, a winter cough being sometimes the forerunner of phthisis.

Emphysema.—A round, barrel-shaped chest, with no alteration in the heart's dullness and no emphysema, is not uncommon in an otherwise normal person. When true emphysema is present the heart's dullness is obscured, and there are the usual changes in the respiratory sounds. I would not pass such a life.

Asthma.—Now and again a history of recurring attacks of spasmodic asthma with no intermediate trouble is obtained. Many people are none the worse for the attacks, and are long-lived. It is often a family failing, and may be an evidence of a gouty inheritance. The life prospects of such an individual of otherwise unexceptionable condition are denoted by the family history generally. When attacks of asthma are associated with emphysema and chronic bronchitis, such people are not insurable, although they often live to their three score years and ten.

Nervous system.—Organic disease of the nervous system is very uncommon in life insurance examinations. Functional disturbances, on the other hand, are common; one of them, the rapid, apprehensive or insurance heart (p. 200), frequently causes a good deal of trouble. A simple form of functional nervousness is the inability to pass water in the presence of the referee, and especially into an unusual receptacle. This can generally be got over by the medical man leaving the room and letting an ordinary bedroom chamber be used.

The rapid heart has already been described. There is also the rare type of man who becomes faint after the examination is all over. All these nervous people, if otherwise unexceptionable in personal history, health, and heredity, are fair risks at ordinary rates.

In dealing with nervous people with tachycardia, the possibility of Graves's disease, especially in an early stage, must be borne in mind, and the throat and eyes carefully examined. When the physical signs are not conclusive, any occurrence of morning diarrhoea should be investigated, because this symptom is not uncommonly present in early Graves's disease.

Pupils.—In every case, whether there is a history of venereal disease or not, the pupils must be examined very carefully, and any abnormalities of size, shape, or movement noted and fully described: as must be the reaction to light and accommodation. Nystagmus must also be looked for. Permanently contracted pupils must not be overlooked when the examination is made in a sunny room. Sluggishly acting pupils probably mean some central nervous lesion, and probability changes to certainty when there is inequality in size. The condition of the knee-reflexes will help the diagnosis. If there is any doubt, a Wassermann test should be insisted upon, whether there is a history of previous venereal disease or not.

Knee-jerks should always be tested, whether there are any pupil abnormalities or not. They will often be found unusually active in nervous persons in insurance examinations. If there be a pathological cause for the change, other signs, such as extensor tendon reflex, ankle-clonus, or nystagmus, will be present. A brisk knee-jerk on both sides with no other abnormal reflexes or pupil changes has no significance.

Absent knee-reflexes almost invariably mean refusal of a policy, and very sluggish ones, suggesting tabes dorsalis, call for the greatest care in examination. The condition of the pupils will help in the diagnosis. Inequality of the knee-jerks suggests general paralysis of the insane, in which case the condition of the pupils will be important.

Mouth.—The tongue, mouth, and fauces should always be examined, although no question is asked about them in many insurance forms. Smoker's throat will often be found, and need not affect the proposal adversely, unless there is other evidence of tobacco excess, e.g. an irregular heart. Such conditions as leukoplakia, and other more certain evidences of syphilis, and tremor in an alcoholic person or a general paralytic, may also be present.

The state of the gums may be important, especially if there is evidence of pyorrhœa, sponginess, or bleeding.

The actual condition of the teeth themselves usually gives no information of value as to prospects of longevity, but a well preserved set suggests good health. If the teeth are nearly all decayed in persons of poor physique and nutrition, or are the seat of pyorrhœa, I would advise deferring the issue

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of a policy until the mouth has been put in a healthier condition and efficient artificial teeth are worn.

The **abdominal organs** should always be palpated, even in the absence of a history suggesting a lesion there, because from time to time some unexpected abnormal condition may be found. Displaced kidneys are occasionally palpated, and with a lax abdominal wall the liver may easily be felt. With an otherwise clean report neither of these conditions need cause any trouble. If there is evidence of immoderate alcoholic consumption a palpable liver may have a serious significance.

A history of a short-circuiting operation for gastric or duodenal ulceration is not at all common in insurance proposals, in spite of the modern frequency of these operations. I would not recommend acceptance unless the scars were linear, sound, the life otherwise unexceptionable, and twelve months had passed without symptoms since the operation. Some surgeons think six months a sufficiently long period of probation.

The War has provided a number of proposals with a history of injury, operation, and war illnesses, and in such cases policies have to be issued with caution. My custom is to report fully on the condition, and give my own impressions as to the likely effect on future health. The final decision rests, of course, with the head-office official.

Urine.—The examination of urine must be carried out carefully and thoroughly. The specimen must, whenever possible, be passed in the examiner's presence. If not, the reason why this was not done should be stated in the medical form, and the guarantee of the proposer that the specimen he sends in is his own should be obtained. There is also the question of orthostatic albuminuria, in which the urine passed on getting out of bed is generally quite free from albumin, while that passed after the erect posture has been assumed contains it. A case of orthostatic albuminuria could thus pass as a first-class life if the morning urine were sent by a "nervous man." Again, the possibility of slight alimentary glycosuria appearing after food must be borne in mind. Urine secreted during the night on a hungry stomach might contain no sugar, and be submitted by a sharp person. At the best there is a good deal of luck in securing urine for examinations at the most favourable time, especially in cases of glycosuria.

The *specific gravity* is generally asked for in

medical reports, and it is useless to report on one below 1010. The examination is often made soon after a meal with which a fair amount of fluid, not necessarily alcoholic, has been taken. This, with the nervous apprehension caused by the visit of the doctor, results in the passing of a urine of specific gravity only a little higher than that of water. In such a case the applicant should be told to go away, empty his bladder completely at once, and return in an hour or more. A suitable specimen will then almost certainly be obtained. With a very low specific gravity the possibility of an attempt to dilute a small amount of albumin or sugar, which is known to exist, must be borne in mind. With a specific gravity above 1030 the possibility of glycosuria must be suspected, and if no sugar is found the head office generally likes an explanation of the high record, such as a hot day, unusually thick clothing, free perspiration, the taking of only a small amount of fluid, etc.

The *reaction* of the urine is asked for by some companies. It is almost invariably acid or neutral, the conditions causing alkalinity being serious enough to attract attention in other ways.

Its *appearance* may attract attention. It may be cloudy when passed, on account of the harmless presence of a considerable quantity of phosphates; but cloudiness may also be due to pus from cystitis, when it means refusal of insurance.

Shreds about half an inch long, or flakes of muco-pus, found in the urine of elderly men come from the prostate, but, unless other symptoms of prostatic disease are present, may not indicate any threatening condition. In younger people they suggest gonorrhoea; careful inquiry, and investigation of the urethral discharge for gonococci, must be made. In all cases, old or young, the question of gonorrhoea must be considered. If gonococci are found the proposal should be declined.

Albumin.—The most delicate and useful test is boiling, after acidulation if necessary. After boiling and acidulation, a faint precipitate is often met with in the urine of pale city clerks of the late 'teens or early twenties. It is doubtful whether this is of any pathological significance. I then test with nitric acid in the cold. Usually there will be no ring at the junction of the fluids, and I report accordingly, recommending for acceptance if, as is generally the case, there is no other abnormality present and the pulse-tension is normal. Some com-

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panies require a microscopical examination for casts before accepting these cases.

A faint trace of albumin with increased pulse-tension in the adult may mean granular kidney, especially in urine of low specific gravity (1010). A history of nocturnal polyuria should be sought for, and will often be obtained. In many cases of high tension, however, no trace of albumin can be found even by the most delicate test. This condition is one of toxic high blood-pressure, and is as uninsurable as that caused by granular kidney.

A thin ring of albumin may be found with the nitric-acid test during a trivial and transitory cold. It is not present when the subject recovers from the cold, and it has no pathological importance.

The albumin of nephritis is present in the urine passed at all times of the day and night, whereas the albumin of functional causes is not found in that passed after a night in bed. Every insurance company looks with suspicion on albuminuria, and will not run any risk when nephritis is the cause. Many refuse cases of functional albuminuria, even if the amount of albumin is slight; a certain number of such cases take on a more serious form, and it is impossible to distinguish them from the others.

Sugar.—It is not at all uncommon in insurance work to find sugar in varying amounts in the urine quite unexpectedly in otherwise unexceptionable proposals. The specific gravity may be high, 1030 or more, or, when there is only a small amount of sugar, 1025 or less. I always add an equal amount of urine to the Fehling solution and boil for twenty seconds. When examining suspected cases after test meals I use twice the bulk of urine to the Fehling solution, and check any precipitate with the phenyl-hydrazin and fermentation tests. The result is invariably confirmed by these tests when a slight yellow precipitate is obtainable with the double quantity of urine to that of Fehling solution.

Sugar will appear once in the urine of some people in an erratic, inexplicable way, and will not be found again in several subsequent examinations extending over weeks or months with unrestricted diet. Such cases are, in my opinion, insurable. In other people sugar will appear regularly after the consumption of a meal of carbohydrates and sugar and a sweet wine, such as port, or sweet grapes, or after one meal in the day only. All are, in my opinion, uninsurable, because some of them

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become actual diabetics before long, and no one can say which will do so.

From time to time it happens that, when a proposal is declined on account of glycosuria, the proposer goes to his own doctor some days afterwards, and no sugar is found. The proposer is naturally dissatisfied and asks for another examination by the medical referee. In such cases I ask the proposer to have a meal comprising in part carbohydrates, sugar, and, if he takes it, some sweet wine, and to come to me two hours afterwards. If no reduction occurs with twice the bulk of urine to Fehling's solution, in each of a series of such examinations spread over some weeks, and the proposal is otherwise unexceptionable, a policy may be issued at ordinary rates or with a few years added. If sugar appears after such a test meal, and especially when the diet has been restricted, the proposal should be declined.

No insurance company likes a proposal with even a small amount of sugar recorded, because it is impossible to tell from one examination what the phenomenon portends.

E. M. BROCKBANK.

LIGHT TREATMENT.—The therapeutic use of light rays is founded upon (a) various experiments showing their stimulating action on animal and vegetable tissues, (b) the power of light to set up inflammation of the skin, as in erythema solare, which has been shown to be due to the actinic rays, (c) the bactericidal and penetrating effects of concentrated actinic light. The late Prof. Finsen, of Copenhagen, revived the ancient method of treating smallpox by red light and the exclusion of ultra-violet rays, and was the first to employ the chemical rays of light in the treatment of lupus vulgaris and other diseases of the skin. For the latter purpose he utilized either the sun's rays concentrated by means of biconvex lenses and filtered through a solution of copper sulphate contained between them, or the light from an arc lamp which is rich in violet and ultra-violet rays. The original *Finsen lamp*, which is still used in large institutions, consists of four tubes or telescopes suspended around a powerful arc lamp, each containing a series of quartz lenses so arranged as to bring the light to a focus. The tubes are water-jacketed, and the space between the lower pair of lenses contains distilled water. The light is thus cooled as it emerges from the tube. Quartz compressors of various shapes are pressed on the skin to render it anæmic;

LIGHT TREATMENT

these also contain circulating water to cool the concentrated rays, and are fixed by rubber bands on the area to be treated or are held in position by a nurse. The small *Finsen-Reyn lamp* is the same in principle as the larger apparatus, but has only one concentrating tube.

The method of procedure is the same for both lamps. A small area of the affected skin having been cleansed with an antiseptic lotion and marked with an aniline pencil, the patient is placed on a couch or chair and the compressor either strapped on by means of elastic bands or held by the nurse during the one hour's exposure. The patient is so placed that the face of the compressor on which the focused rays impinge at a right angle is just within the focus of the cone of light. The duty of the nurse is to see that the patient does not move and that steady pressure is maintained on the skin. About twenty-four hours after the application an inflammatory reaction, sometimes accompanied by the formation of a bleb, occurs, and is dressed with a simple antiseptic ointment. The applications are repeated daily, or, if necessary, two or more are given in one day. Owing to the small area of skin exposed, and the necessity for frequent repetitions of the treatment to a given area, the method is apt to be extremely long and tedious, and in extensive and rapidly spreading cases of lupus the application of a caustic preparation, such as pyrogallie-acid ointment, or some other method such as X-rays, should be combined with the Finsen light. The final results of the treatment are, however, extremely satisfactory, and the scar is better than that resulting from any other method. The Finsen light is most successful and has been most used in treating lupus vulgaris, but it has also been employed with some success in cases of alopecia areata, lupus erythematosus, capillary naevi, rodent ulcer, etc.

Other lamps are those of *Lortet* and *Genoud* and *Marshall & Woods*, in which the rays are not focused but the patient is brought close to the source of the light; and the *Bang*, *Miller*, and *Dermo* lamps, in which one or both electrodes consist of iron and yield a high percentage of ultra-violet rays. The last have been superseded by lamps with tungsten electrodes. In the *Kromayer* and *Cooper-Hewitt* lamps the actinic rays are produced by mercury vapour contained in quartz tubes and raised to a high temperature. In all these lamps larger areas can be treated, and the exposures required are shorter, than in the case of the Finsen lamp, but it is doubt-

LIGHTNING, ETC., DEATH FROM

ful if they are so efficacious in deep-seated diseases such as lupus vulgaris; they are, however, of value in the treatment of most of the varieties of alopecia and some chronic, superficial scaly diseases such as psoriasis and chronic eczema, and in septic wounds and ulcers and some cases of boils and pustular acne. Tuberculous peritonitis has also been treated with good results.

Light baths come into a different category, as their therapeutic action depends for the most part upon dry heat, which can be raised to a higher temperature with less discomfort to the patient than moist heat. The patient is placed in a cabinet which has internal reflecting surfaces and contains a number of incandescent electric light bulbs by which the air is raised to a high temperature, an aperture being provided in the lid of the bath for the patient's head. Local baths for a limb or other part of the body may also be used. In a few light baths small arc lamps are employed, or a large searchlight is played upon the patient's body. In this way some benefit may result from the actinic rays. Light baths are generally employed for their general effect and to replace hot moist-air baths in rheumatic and allied conditions. Sun baths have been very little used in this country; they are now being employed at Alton by Sir H. Gauvain in the treatment of surgical tuberculosis with considerable success. It is doubtful if coloured-light baths possess any therapeutic value apart from the heat generated.

S. E. DORE.

LIGHTNING AND ELECTRIC DISCHARGES, INJURIES AND DEATH FROM.

—Death from lightning is not infrequent in this country, and the effects produced may resemble closely those resulting from mechanical violence.

The lesions produced are very various. Bruises, lacerations, fractures (often multiple) of almost every description, and burns may be met with. Arborecent markings resembling Lichtenberg figures are sometimes found on the skin and are characteristic. The clothes are usually torn and may be singed, and, while they may even be torn to shreds, the parts of the body covered by them may show no signs of injury. Rigor mortis has been noted in numerous cases. When a body found dead after a thunderstorm shows no marks of violence, it is well to remember that the cause of death may have been syncope from fright.

In non-fatal cases the person is either partially stunned or immediately rendered unconscious. The muscles are relaxed, the pulse is soft and slow, the breathing slow, and the pupils are dilated and reacting to light. Delirium, blindness, deafness, and paralysis may also occur. Temporary affections are probably due to nerve shock, and permanent paralysis, blindness, or deafness to actual lesions of nerve-tissue. In those cases in which there is hope of resuscitation, treatment consists in performing artificial respiration and the adoption of general measures to stimulate cardiac action. Injuries are treated *secundum artem*.

Cause of death.—Death may result from the extent or nature of the injuries—e.g. fracture of skull, or wounds of great vessels—or from nervous shock when gross injuries are absent. It should be understood that whereas the conducting power of muscle is fifteen million times weaker than that of copper, nerve-tissue conducts almost as well as metal.

Post-mortem examination.—There are no appearances actually characteristic. Lesions of the nature already described may be present, or no injury (external or internal) may be found, death in these cases being due to direct action on the nerve-centres. When effusion of blood has taken place into the cranial cavity or chest, it has been found to be associated with lesions of adjacent parts. While it has been stated that the blood is abnormally fluid, cases have been recorded in which clotting has taken place as usual.

Electricity.—Deaths from electric shock increase in number year by year owing to the increased use of electric power for industrial purposes. The usual cause of death is arrest of cardiac action, but primary arrest of respiration may occur. The actual cause will depend on the points of entrance and exit and the path of the current. Alternating are more dangerous than constant currents. The voltage which is capable of producing death varies in differing circumstances. Whereas voltages under 250 have in some cases proved fatal, recovery has followed a shock from 2,500 volts. The dryness or otherwise of the hands and clothing is a factor of importance, as the conductivity of the body is increased if the clothing or hands are wet.

The extensive lesions found after lightning-stroke are not met with here. No lesions may be present, or burns of varying degree may be found, due to contact of the skin with a live wire, or to the clothing having taken fire.

Burns are sometimes found on the body when there is no evidence of burning of the clothing covering the parts. Electric burns, as distinguished from burns produced by flame, are dry and aseptic, and vital reaction in surrounding tissues is slow in appearing. If a live wire has been the cause of the burns, greenish staining due to the fusion of the copper may be met with.

Accident, suicide, or homicide?—This question will not usually arise. The cases are almost all accidental. Electrocutation is the method adopted in the United States of America for inflicting the ultimate penalty of the law. A case of suicide by taking hold of the conductors of a dynamo machine has been reported (*B.M.J.*, 1885, i. 550).

A. ALLISON.

LIPODYSTROPHIA.—Symmetrical disappearance of fat from the subcutaneous tissue of the upper part of the body. The condition is rare. The first noticeable feature is that the face becomes gaunt and senile from loss of adipose tissue, so that the eyes are sunken, malar and other prominences prominent, and the muscles and vessels conspicuous. (Fig. 50.) Gradually the neck becomes affected, and then the absorption of fat spreads gradually downwards, implicating upper limbs, thorax, and abdomen, but never extending beyond the pelvic girdle. The buttocks and legs, for this reason, appear unduly massive. There is no pain or other sensory change, and no other structure is affected, the muscular power remaining as good as ever. Females are more prone to the disorder than males. The commonest age of onset is about 5 years. The only disability is the altered appearance, which, simulating that of emaciation, may suggest the existence of tuberculosis, new growth, or diabetes. A more careful examination shows that the loss of fat is bounded by the pelvic girdle, and is not accompanied by muscular weakness. No treatment is known.

FREDERICK LANGMEAD.

LIPOMATOSES.—This group of diseases is of interest from an academic rather than from a practical point of view. The relation that appears to exist between the nervous system and the formation of masses of subcutaneous fat suggests various speculations which unfortunately have not as yet led in the direction of successful treatment.

Most striking of all the types of lipomatosis

is that known as *adiposis dolorosa* (q.v.). Similar to it in many respects is that sometimes labelled *nodular circumscribed lipomatosis*, a not uncommon disorder. It is characterized by the formation of lipomatous masses, small rather than big, often symmetrically arranged upon the limbs and trunk, and sometimes tender and painful. This may be associated with a diffuse fibromatosis of the skin. Most often the subjects are middle-aged women, and there may be psychical symptoms. Treatment is called for as a relief to the pain and tenderness. As might be expected, it is not very easy to get rid of these troubles. Anæsthetic liniments, e.g. oily solutions of menthol, give the best results. The pain may disappear spontaneously, even after months or years.

Between this and the curious *diffuse lipoma of the neck* there are not many points in common. The latter disorder is one affecting stout middle-aged men. Some writers connect it with chronic alcoholism. A group of lipomata, more or less symmetrically arranged, develops in the nape of the neck, and increases in bulk to form a thick collar of fatty tissue, usually painless, but disfiguring and inconvenient in the extreme.

Surgical treatment is not very satisfactory owing to the widely ramifying distribution of the growth.

For these and similar conditions thyroid extract is sometimes given, but it must be confessed that the impression made is small.

CAREY COOMBS.

LIPS, AFFECTIONS OF.—The affections of the lips to be considered in this article may be grouped under three headings, viz. —Deformities, Inflammatory Conditions, and Affections of Lip Movements.

DEFORMITIES

Macrostoma (congenital enlargement of the buccal fissure) and *Microstoma* (congenital smallness of the buccal fissure) both occur. The latter may need surgical interference.

Macrocheilia is a swelling of one or both lips, usually the lower only, associated with hyperplasia of the connective tissue, and an angiomatous condition of the lymphatics; in some cases the blood-vessels also are hypertrophied. Treatment is surgical.

A moderate degree of lip enlargement is seen in other conditions, e.g. as a result of inflammatory reaction of the tissues around recurring ulcers in unhealthy children; thickening and eversion is common in cretins and other mentally defective children.

Hare-lip is considered in a separate article under that title.

INFLAMMATORY CONDITIONS

Fissures occur frequently in debilitated patients, especially in children; they affect usually the lower lip

and are a common result of "chapping" from exposure to cold and dry air. Fissures at the angles of the mouth in infants and young children are often evidence of congenital syphilis; such ulcers are surrounded by a red area, are painful, heal slowly, and lead to

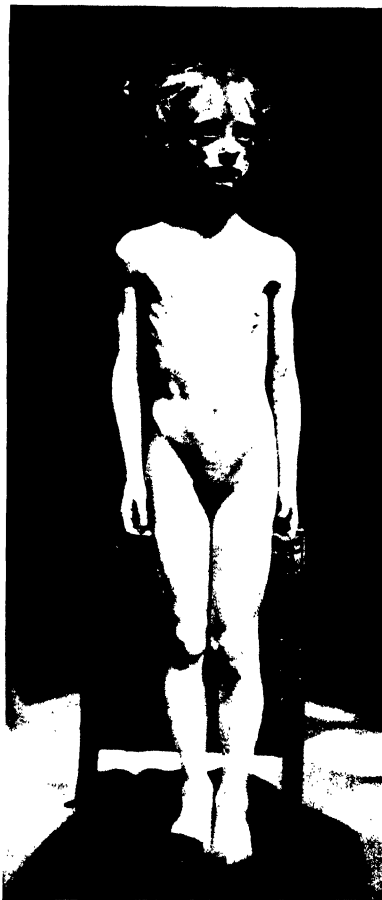


Fig. 50.—Lipodystrophia.

LIPS, AFFECTIONS OF

permanent scars radiating from the angles of the mouth (rhagades). Treatment of fissures includes the use of antiseptic salves locally, general measures to promote health, and, in the syphilitic cases, active specific medication.

Papillomata and *retention cysts* of sebaceous or mucous glands are occasionally seen on the lips.

Impetigo, *herpes*, and *farunculosis* may affect the lips, but are described under their respective headings.

Sordes, often seen in exhausting and febrile diseases, is due to the accumulation of epithelial debris. The lips should be sponged frequently with weak antiseptic lotions, and the surface kept soft by non-irritant ointments such as lanolin.

Tubercle affects the lips in two ways—(a) as lupus vulgaris, usually by extension from the cheek; (b) as a solitary tuberculous ulcer of slow growth, with thickened edge and irregular floor. This ulcer, which is often painful, is almost always associated with tuberculous lesions of the lung or larynx. Such an ulcer may yield quickly to X-ray treatment; failing this, surgical measures are indicated.

Syphilis may affect the lips in any stage. *Primary chancre* is fairly common, especially upon the upper lip and in young girls. Contact-chancres on both lips may occur. Lip chancres resemble other extragenital chancres in being softer, moister, and leading to more local inflammatory swelling than the genital variety. The submaxillary glands are involved early. The chancre begins as a hard plaque below the surface, which soon ulcerates and breaks down. As a rule the condition is painless, unless septic infection has occurred. The history of the case, the age of the patient, the absence of tuberculous lesions elsewhere, the presence of early glandular enlargement, and the occurrence of secondary syphilitic signs usually suffice to establish the nature of the lesion. Confirmation may be obtained by finding the spirochete in scrapings from the surface.

Secondary syphilis may produce mucous patches upon the lips similar in nature to condylomata about the anus. (See SYPHILIS.)

Tertiary syphilis affects the lips in two ways. The more common is a gummatous ulceration which has to be distinguished from a tuberculous lesion; but it is usually larger, painless, is associated with other syphilitic mani-

festations, and other foci of active tuberculosis are absent; solitary tuberculous ulceration of the lip is a rarity. In the other type of syphilitic manifestation the lip may become enlarged and hypertrophied by connective-tissue overgrowth, comparable to the periosteal thickenings common upon the tibia. This condition is rare.

Congenital syphilis frequently affects the lips of infants and young children in the form of shallow ulcers, as noted in connexion with fissures (see above). In later childhood destructive ulceration similar to that which affects the alae nasi or cheek may occur upon the lips.

Epithelioma, beginning as a fissure, wart, or submucous nodule, is not uncommon in later middle life; it is almost confined to men, and almost invariably affects the lower lip. The influence of smoking, especially through short and hot pipe-stems, is important. The growth arises at the junction of skin and mucous membrane, and extends with moderate rapidity. In whatever form the process commences, painful ulceration is liable to occur early. Local swelling of the lip is not a pronounced feature. The submaxillary glands are affected early, but in sequence—not, as in chancre of the lip, in groups. The only satisfactory treatment is early and complete removal. If the case is inoperable, pain may be greatly relieved by X-rays or radium.

AFFECTIONS OF THE LIP MOVEMENTS

A *unilateral paralysis* of the lips occurs in lesions of the corresponding facial nerve. Unless the nerve is completely divided by accident or middle-ear disease, the prospect of ultimate recovery is good. Hemiplegic paralysis of the face is very rarely permanent.

Bulbar palsy, due to degeneration of the bulbar nuclei of both sides, leads to paralysis of the orbicularis oris, and consequent inability to close the lips and retain saliva: its association with palatal, lingual, and pharyngeal paralysis suggests the correct diagnosis. The condition is incurable.

Pseudo-bulbar palsy, due to double hemiplegia from lesions of both internal capsules, is rare; the suddenness of the onset is in striking contrast to the true bulbar form.

Myopathy of the Landouzy-Dejerine type in adolescent males includes in its symptomatology labial weakness leading to prominence of the lips and difficulty in closing the mouth.

LIVER, ABSCESS OF

The facial expression which results has been compared to that of a tapir.

Tremor of the lips is an early and valuable sign in general paralysis; it also occurs in delirium tremens, but is rarely seen in paralysis agitans.

C. E. SUNDELL.

LIPURIA (see URINE, EXAMINATION OF).

LITHURIA (see URINE, EXAMINATION OF).

LITTLE'S DISEASE (see CEREBRAL DIPLEGIA).

LIVER, ABSCESS OF.—Multiple hepatic abscesses result from the action of pyogenic organisms which reach the liver by one of its afferent or efferent channels. The organisms may be brought by the blood (1) through the arterial system, as in general pyemia from any cause; (2) by the portal vein, in septic thrombosis of that vein, or from a source of suppuration in the area which it drains: thus the infection may be derived from a suppurating or gangrenous appendix, peritonitis, intussusception, and many other lesions; (3) by the umbilical vein, in the newly born, in umbilical sepsis. On the other hand, the abscesses may occur about the biliary tributaries, and be secondary to suppurative cholangitis of the larger ducts. Multiple abscesses are of pathological rather than clinical importance, and are sufficiently described in the articles on the conditions with which they are associated.

Single abscesses occasionally result from those causes which more commonly lead to multiple abscesses. They may also be due to the secondary invasion, by pyogenic bacteria, of such local conditions of the liver as actinomycosis, hydatid cyst, tuberculosis, or neoplasm. Tropical abscess falls into a different category (see TROPICAL ABSCESS). FREDERICK LANGMEAD.

LIVER, ACTINOMYCOSIS OF (see ACTINOMYCOSIS).

LIVER, ACUTE YELLOW ATROPHY OF.—A form of degeneration and necrosis of the liver, due to toxins, and producing a profound auto-intoxication.

Acute and subacute yellow atrophy of the liver are due to toxins which cause necrosis of the liver-cells. Acute yellow atrophy produces a diminution in the size of the organ, but in subacute atrophy it may become larger than normal. The cerebral and other severe symptoms are due to auto-intoxication.

Etiology.—The disease, especially the acute

LIVER, ACUTE YELLOW ATROPHY OF

form, is commonest in young adults, but may occur at any age. Subacute atrophy is the commoner form in children. Females are more often affected than males, owing to the association of the disease with pregnancy.

Two factors appear to be concerned—(1) a toxin which attacks liver-cells in particular, (2) a liver incapable of dealing with toxins to the normal extent.

The toxins are either chemical or microbic, and all act to a varying extent in three ways. They cause fatty degeneration and necrosis of liver-cells, resulting in auto-intoxication; cholangitis of the small bile-ducts; and increased viscosity of the bile. Some cause increased blood destruction in addition.

Examples of chemical toxins are trinitrotoluene, tetrachlorethane, arsenobenzol derivatives, chloroform, phosphorus, and arseniuretted hydrogen.

Among the more important microbic poisons are those of syphilis, spirochaetosis icterohæmorrhagica, infective and catarrhal jaundice.

It is possible that poisons produced in pregnancy may be sufficient alone to cause the disease, but undoubtedly many of the cases are due to the toxins enumerated acting on a liver already weakened by dealing with the poisons elaborated in all pregnant women.

Pathology.—In acute yellow atrophy the liver is greatly shrunken, soft, and smooth. The colour on section is yellowish with red areas scattered throughout its substance. The lobules can be recognized no longer. In sub-acute cases it is often larger than normal.

When life is prolonged sufficiently, attempts at repair take place. The most necrotic parts become extremely fibrosed, and the less damaged cells recover and proliferate irregularly, forming nodular swellings often called adenomata.

It can be seen under the microscope that, in the red areas, where necrosis is most advanced, the liver-cells have disappeared and nothing but stroma and capillaries remain. In the less affected parts the liver-cells show necrosis or fatty degeneration. In subacute atrophy most of the cells are necrotic or degenerated. The bile-ducts show proliferation of the cells lining them, which causes the obstructive jaundice. If the condition lasts long enough there is evidence of irregular proliferation of liver-cells, many of which are unduly large and show mitosis, and of proliferation of bile-ducts.

LIVER, ACUTE YELLOW ATROPHY OF

Symptomatology.—At first the symptoms are those of the special condition which causes the toxæmic jaundice. Then the liver becomes progressively and often rapidly smaller. In many cases enlargement of the spleen follows. Increasing cholangitis causes deeper jaundice, but one or two cases have been recorded with no jaundice. Hæmorrhages occur from the nose, stomach, bowel, or under the skin. The urine contains bile pigment, albumin and casts, and leucine and tyrosine. Vomiting and diarrhoea, followed by delirium, coma, and, in some cases, convulsions, set in.

Less severe cases may live for many months, only to die of cirrhosis; but occasionally recovery takes place.

Diagnosis.—The most important signs are diminution in the size of the liver, with increased severity of the constitutional symptoms. Deepening jaundice, persistent vomiting, and especially hæmorrhages occurring at a period when the primary illness should be abating, are warning signs. The appearance of albumin and casts in a urine previously free from them is a valuable help, and the presence of leucine or tyrosine is almost pathognomonic. Cerebral symptoms arise near the end of the illness.

Prognosis.—The most acute cases all terminate fatally, and even in subacute cases the mortality is high. Death may take place as early as the second day, but is most frequent in the second and third weeks.

Treatment should be started at the first sign of this grave disease. The diet should be fluid, fat-free, and contain little protein. Dilute skimmed milk with the addition of dextrose is sufficient. Saline aperients must be given to eliminate toxins. Vomiting can sometimes be checked by dilute hydrocyanic acid (3 or 5 min. to a dose), or by tincture of opium as a palliative measure, but as soon as possible sodium bicarbonate 3 dr. and glucose 4 dr. to half a pint of water should be given by the rectum eight-hourly, or sodium bicarbonate 2 dr. to a pint of normal saline subcutaneously or intravenously twice a day. Subacute cases have made excellent recoveries with this treatment.

E. A. COCKAYNE.

LIVER, AMYLOID DISEASE OF (see AMYLOID DISEASE).

LIVER, CHRONIC CONGESTION OF (see LIVER ENLARGEMENT, DIFFERENTIAL DIAGNOSIS OF).

LIVER, CIRRHOSIS OF

LIVER, CIRRHOSIS OF.—The term cirrhosis, which originally referred to the yellow colour of the liver, is now used for any form of fibrosis of the organ. The chief forms are—

Pericellular.

Portal or multilobular.

Biliary.

More than one form may be present at the same time, causing a mixed cirrhosis.

PERICELLULAR AND PORTAL CIRRHOSIS

Etiology.—Secondary syphilis, especially the congenital form, is the most important cause of **pericellular** cirrhosis, and tertiary syphilis causes gummatous cirrhosis. These are considered under LIVER, SYPHILIS OF.

Portal cirrhosis is due to one of two factors—(1) the action of some toxin, chemical or microbial, conveyed by the portal vein or manufactured in the liver itself; (2) a deficiency of power, congenital or acquired, on the part of the liver-cells to destroy toxins—or, very rarely, to mechanical irritation by the ova of a parasitic worm.

Of the chemical poisons, by far the most important is alcohol. It is, however, generally accepted that ethyl alcohol is not the direct cause of the disease, though some observers think that the higher alcohols, such as amyl alcohol or potassium sulphate and bisulphate, which are present in wine and beer, may cause it by their direct action. The general opinion is that alcohol produces fatty degeneration of the liver-cells and so renders them susceptible to the action of alimentary toxins, and that by causing chronic gastro-enteritis it allows the formation of alimentary toxins in excessive amounts and renders the mucosa more permeable to them. Constitutional incapacity of the liver to destroy toxins undoubtedly plays an important part in this, as in all forms of portal cirrhosis.

The disease is far commoner in alcoholic than in temperate subjects, and is three times as frequent in males as in females. Certain occupations are highly favourable to its development, such as those of the publican and commercial traveller. The frequency of its occurrence in different countries does not run parallel with the amount of alcoholic liquor consumed in them. It is much commoner in England than in Scotland, and in temperate regions than in the tropics. This is accounted for by the indirect and complex action of alcohol.

Chemical poisons used in the manufacture of munitions during the War have given rise

LIVER, CIRRHOSIS OF

to cases of cirrhosis, and for many years we must bear this in mind as a possible cause in cases of obscure etiology. Examples of these poisons are trinitrotoluene and tetrachlorethane. Organic and inorganic compounds of arsenic also can cause the disease.

Microbic poisons fall into two main groups. In the first are those produced in the course of any long-continued gastro-enteritis. Apart from alcoholic gastro-enteritis, this is commonest in the East, especially in India amongst young Mohammedans, Brahmins, and Hindus, owing to their fondness for hot condiments such as curries, pepper, cardamoms, and ginger, which cause chronic inflammation of the alimentary tract. Microbic poisons of the second group have a specific action on the liver-cells, and the organisms in these diseases are either suspected or known to multiply in the liver itself. Spirochaetosis icterohaemorrhagica, syphilis, relapsing fever, kala-azar, and catarrhal jaundice are examples of these diseases.

It is considered probable, though proof is lacking, that bacteria are sometimes conveyed to the liver by the portal vein or hepatic artery and, multiplying there, cause cirrhosis.

The spleen has been regarded by some as the site of origin of toxins which can produce portal cirrhosis. In many blood infections organisms collect there in great numbers, and their toxins may reach the liver by the portal vein. Malaria has been cited as an example, but cirrhosis is rare in this disease. Splenic anaemia is probably due to a primary infection of the spleen, and recent work has suggested that a streptothrix is the agent. The cirrhosis, which occurs late in the disease, is almost certainly due to poisons derived from the spleen.

Portal cirrhosis in children is sometimes due to alcohol, which is given by ill-educated and intemperate parents to babies or young children to keep them quiet. The etiology in other cases is obscure, but gastro-enteritis plays no important rôle. Syphilis in children, as in adults, weakens the liver, and is well known as an indirect cause of true portal cirrhosis.

The mechanical irritation of the liver due to the presence of ova of *Schistosoma mansoni*, which causes rectal schistosomiasis, of *S. japonicum*, and more rarely of *S. hematobia*, may give rise to a peculiar form of portal cirrhosis in heavily infected persons. It is commonest in Egypt and Japan.

Special forms of portal cirrhosis which deserve separate mention are those found in hæmochromatosis (q.v.) and progressive len-

ticular degeneration (see LENTICULAR DEGENERATION, PROGRESSIVE).

Pathology. Morbid anatomy.—The size of the liver varies greatly. Sometimes it is much diminished, and the organ may weigh as little as 30 oz. This form is often spoken of as atrophic. In other cases it is very large and may weigh 180 oz. Every intermediate size is found. The enlarged cirrhotic livers show a high degree of fatty change in addition to cirrhosis, and are sometimes referred to as hypertrophic—a term which should be restricted to primary biliary cirrhosis. They are commoner in young people.

The surface of the liver is irregular; sometimes the nodules are small and give it a granular appearance, at other times they are much larger and produce the well-known hobnailed form. The nodules are tawny in colour. In cirrhosis due to specific infections or poisons used in the manufacture of munitions, and occasionally in the alcoholic form, there are large nodular areas with very extensive intervening patches of fibrosis. These large nodules are sometimes spoken of as multiple adenomata. In reality they are the seats of active regeneration.

The surface is opaque owing to subcapsular fibrosis, and may be adherent to neighbouring structures.

On section the organ is extremely tough. The cut surface exhibits islands of yellow liver substance, composed of varying numbers of lobules separated by a dense meshwork of greyish fibrous tissue. There is also marked fibrosis of the portal spaces. Sometimes there is thrombosis of the portal vein or its branches, and there may be hæmorrhages in the liver substance or in the fibrous strands.

Histology.—The liver-cells, whatever be the cause of the disease, always show degenerative changes before there is increase of connective tissue. Some cells become necrosed and others, less damaged, divide. This is much more obvious in cases due to trinitrotoluene and similar poisons, and in acute microbial infections than in cases in which the poisoning extends over many years, as in alcoholism. When regeneration has been active, the new hepatic cells are irregularly disposed and larger than normal. They are visible in the larger "hobnails" and in nodular hyperplasia following subacute yellow atrophy.

In the early stages there is round-celled infiltration in the portal spaces and between the groups of lobules; later it is seen in associa-

LIVER, CIRRHOSIS OF

tion with new and old connective tissue in proportions varying according to the duration and activity of the disease.

Regenerative changes take place in the small bile-ducts, so that numerous little blind canaliculi are seen in the fibrous tissue between the lobules and in the portal spaces.

The spleen is often large and fibrotic, especially in children and young persons.

Symptomatology.—In a certain proportion of cases the disease gives rise to no symptoms and is discovered accidentally after death. In the majority the earliest symptoms are loss of appetite, flatulence, nausea, and morning vomiting. Either constipation or diarrhoea may be present at first, or they may alternate. Diarrhoea is sometimes intractable in the latter stages. The complexion becomes sallow, and dilated vessels appear on the cheeks, nose, and conjunctivæ. Spider nævi are common on the face and body. The lips are dry, the tongue is dry and furred, and the pharynx injected. Dilated veins are often present over the upper part of the abdomen and round the umbilicus, where they form the *caput Medusæ*. A line of small dilated venules along the line of attachment of the diaphragm is no indication of hepatic disease.

Hæmatemesis is common, and may be the first sign that the patient is suffering from a serious malady. It is usually copious and often recurrent, but is seldom the immediate cause of death. It arises from the rupture of a dilated vein at the lower end of the œsophagus, or much less often from congestion or superficial erosions of the gastric or pharyngeal mucosa. Hæmorrhoids occur, but do not so often cause troublesome symptoms as might be expected.

The urine is scanty, very acid, and highly coloured owing to the presence of uroerythrin. On standing there is a heavy precipitate of pink urates.

Jaundice, if it appears at all, is slight and often transient. It is a late sign.

In advanced cases the emaciation of the face and body is very striking.

In atrophic cases there is diminution in the liver-dullness. When the liver is enlarged the edge may be felt, sometimes just below the costal margin, sometimes below the umbilicus, and is very hard and sharp. The liver-dullness is increased upwards and downwards, and fine or coarse nodules can be felt on the surface. There is often some tenderness and dull aching pain in the right hypochondrium. An enlarged hard spleen can be felt in some cases, especi-

ally in young subjects, and in the more acute cases. The abdomen is distended. Late in the disease ascites is common.

Intermittent pyrexia may be present in acute cases and in advanced cases of a chronic type.

Epistaxis and oozing from the gums occur late and are due to toxæmia.

The heart-muscle becomes flabby and cardiac dilatation may result. A systolic murmur at the apex is often heard. (Edema of the legs may be caused by heart failure or by ascites.)

Death may be due to single or rapidly repeated hæmatemeses, to heart failure, or to toxæmia. In toxæmia cerebral symptoms, drowsiness, delirium, coma, or convulsions may terminate the illness. Death due to some intercurrent disease, especially to pulmonary or peritoneal tuberculosis, is quite common.

The total duration of the disease is very variable. The most rapidly progressive cases occur in young adults who have been heavy drinkers, or follow subacute atrophy; in such cases death usually occurs within two to six months. In the more chronic cases death generally takes place two or more years after the symptoms are well established.

Diagnosis.—Rapidly progressive cases may resemble *hypertrophic biliary cirrhosis*, for jaundice and fever with enlarged liver and spleen may occur in both; the course of portal cirrhosis, however, is shorter and the jaundice is usually transient. In this country, in a doubtful case a diagnosis of portal cirrhosis is much more likely to prove correct than one of biliary cirrhosis.

Cases with a large liver and spleen but no jaundice must be diagnosed from chronic *malaria*. In the latter there will be a history of attacks of ague; anæmia, which is absent in cirrhosis except after an attack of hæmatemesis, will be present. Low fever is commoner in malaria. A history of temperate habits is in favour of malaria, but must not be trusted entirely. The spleen is generally larger in malaria; and parasites may be found in the blood, though they are often absent in malaria, especially in the old cachectic cases in which enlargement of the liver is commonly found.

Spleno-medullary leukaemia may resemble cirrhosis in the large size of the liver and spleen, but the spleen is usually much larger than in cirrhosis. A blood-count will make the diagnosis certain. To *chronic lymphatic leukaemia* the same remarks apply, except that in this

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disease enlarged lymphatic glands are almost always present and add another distinguishing feature. *Splenic anaemia* is much harder to distinguish. *Hæmatemesis* may occur quite early in this disease, and may recur. The spleen, however, is larger than in cirrhosis, and secondary anaemia with leucopenia is found. Since removal of the spleen is curative in splenic anaemia, the differential diagnosis is of the utmost importance. When cirrhosis of the liver has developed in a case of splenic anaemia and produced the syndrome that is known as Banti's disease, the diagnosis is still more difficult, but much less important. The pronounced anaemia is the most valuable sign in favour of Banti's disease.

From *fatty enlargement* of the liver cirrhosis is distinguishable by the greater hardness, the nodular surface of the organ, and its sharp edge. In fatty enlargement there are no signs of portal obstruction.

Tertiary syphilis is generally easy to distinguish when the liver is palpable, its surface in this disease being so much more irregular. Diagnosis is much more difficult when the liver cannot be felt owing to its small size or to the presence of ascites. But other signs of syphilis are often present, and the Wassermann reaction is positive, though it must be remembered that true multilobular cirrhosis may supervene in cases of syphilis.

Perihepatitis is often confused with cirrhosis. The smooth, rounded, and often everted edge of the liver when it is palpable in this disease is unlike the sharp, irregular edge in cirrhosis. A history of several tappings is much against cirrhosis, and albuminuria is in favour of perihepatitis. Ascites in perihepatitis appears early while the nutrition is good, whereas in cirrhosis it occurs, as a rule, near the termination of the illness.

Some cases of *malignant liver disease* simulate cirrhosis very closely. The nodules on the surface of the liver may be larger and a smooth surface may be felt between them, whereas in cirrhosis the whole surface is nodular. In the larger nodules in malignant disease, umbilication may be felt and so make the diagnosis certain. In new growth, jaundice and anaemia are more common. Its progress is quicker, and rapid increase in the general size of the liver or in the nodules indicates the presence of secondary growths. Careful examination will often reveal the primary neoplasm. Enlarged glands near the left clavicle or at the umbilicus, or secondary growths in other parts

of the body, will establish the presence of malignant disease.

Hydatid disease, deep in the liver, causes enlargement, but the organ is smooth and not so hard as in cirrhosis. The general health is not impaired and the symptoms of cirrhosis are absent, with the exception of aching in the right hypochondrium.

Tropical abscess of the liver may resemble cirrhosis. A history of residence abroad, with or without a definite history of dysentery, can be obtained. Absence of dyspepsia and of alcoholism is in favour of anaemic infection. The liver is softer, smooth, and more tender, and the swelling is often local in tropical abscess. Higher fever and leucocytosis are also valuable signs of this disease.

In *intrahepatic suppuration* due to other diseases the constitutional symptoms and pain and tenderness are still more marked.

The clinical course of some cases of cirrhosis is very like that of *intestinal obstruction*. Just before the onset of ascites there may be vomiting, extreme constipation with severe flatulence, and a history of previous alternating diarrhoea and constipation. In such cases it is usually impossible to feel the liver. The appearance of the patient and his history usually give sufficient help to make differentiation possible.

In cases of *chronic heart disease*, with engorgement due to failure of compensation, the liver may be enlarged and rather hard and there may be ascites. The diagnosis ought not to offer much difficulty, except from cases of cirrhosis with secondary heart failure. When heart failure is primary, the history of dyspnoea and often precordial pain and palpitation is helpful. Edema of the legs in morbus cordis generally precedes ascites, and cyanosis is often a feature. Rheumatic cases occur earlier in life than cirrhosis, and there is cardiac enlargement with well-marked murmurs due to valvular disease.

In *chronic tuberculous peritonitis* there may be enlargement of the liver and spleen and ascites, but this disease occurs in young people, as a rule, and a thickened omentum can generally be felt. The facies is unlike that of cirrhosis.

Enteric fever may resemble the acute form of cirrhosis of the liver in having fever, diarrhoea, and a large spleen. The liver is usually palpable in these cases of cirrhosis, the spleen is larger, rose spots are absent, and the temperature is lower. A positive Widal

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reaction is present in enteric fever at the period when diagnosis is difficult.

Kala-azar, which is endemic in the Mediterranean area and the East, may be mistaken for cirrhosis, but the spleen is generally larger, and pyrexia is present at the beginning of the illness and recurs periodically. Splenic puncture, an operation by no means free from risk, will reveal the presence of Leishman-Donovan bodies.

The absence of persistent jaundice distinguishes portal from *obstructive biliary cirrhosis*.

Early cases of *hamochromatosis* may be mistaken for ordinary portal cirrhosis, but when it is well established the peculiar bronzing of the skin is quite sufficient for differential diagnosis, even in the absence of glycosuria.

If sugar is present in the urine later than three hours after the administration of 40 grm. of galactose, there is serious damage to the liver function, and diseases such as simple splenic anæmia, perihepatitis, tumours, hydatids, chronic congestion, and enteric fever can be dismissed. A very early indication of hepatic insufficiency is the production of a deep-rose colour on adding two drops of Ehrlich's aldehyde solution to 5 c.c. of urine. This is especially valuable when the symptoms are suggestive of cirrhosis but physical signs are absent.

Prognosis.—Portal cirrhosis is nearly always progressive and fatal, though in some cases there is arrest of the fibrosis and sufficient compensatory hyperplasia takes place to maintain health. It is more rapid in young people than in old.

A large spleen is of bad import and often indicates the imminence of hæmatemesis or ascites. Ascites usually presages early death, and jaundice, fever, and oozing of blood from the gums are of evil significance. (Edema of the legs is also a bad sign. Increasing emaciation is unfavourable. On the other hand, a small liver and spleen with well-defined caput Medusæ indicates that the collateral circulation is good and enables a better prognosis to be given.

Treatment.—During the earlier part of the disease the most important consideration is to prevent further ingestion of toxins. In all cases alcohol should be prohibited entirely, nor should alcoholic tinctures or chloroform water be prescribed. When the nature of the occupation is such as to make the refusal of alcohol difficult, the patient should be persuaded to change it. Removal from the influ-

ence of injudicious friends should, if possible, be secured. Irritating foods such as curries, pickles, and pepper must be avoided. In cases due to chemical poisons absorbed in carrying on a dangerous trade, the acute stage of the illness will have necessitated its being given up, and it must not be resumed. Arsenic is contraindicated.

Milk diet is necessary in the more acute cases and in the late stages of more chronic ones. When milk is not tolerated, buttermilk or skimmed milk must be substituted.

Fats and proteins are not dealt with satisfactorily by the damaged liver, so that fats ought to be restricted and proteins given in the form of boiled fish, eggs, and boiled chicken. Sugars are indicated for the liver disease, but the chronic dyspepsia may make it impossible to give much carbohydrate food owing to the fermentation produced. As much should be given as the dyspepsia permits.

Iodides are supposed to promote the absorption of the excess of connective tissue. They should be given a trial, especially in view of the fact that syphilis is an indirect cause of portal cirrhosis in some cases. The value of iodides is probably based on their good effect in cases of this class.

For the *gastritis*, sodium bicarbonate with infusion of gentian before meals is useful; it also aids in combating acidosis. Bismuth compounds, especially the salicylate, may be employed with advantage to some patients, especially when flatulence is troublesome. In cases with atony of the stomach the following prescription is effective:—

Ry Liq. strych. ℥iij.
Acid. nitro-hydrochl. dil. ℥x.
Inf. aurant. ad ʒi.
T.d.s., p.c.

For *constipation*, morning salines or aperient waters such as Apenta, Hunyadi János with meals, and a blue pill or vegetable laxative at night, are the best. Alkaline waters such as Vichy and Apollinaris are good. Moderate diarrhœa ought not to be checked, but, if it be excessive, bismuth and opium, or tannigen, will often control it.

Slight attacks of *hæmatemesis* due to oozing from the congested gastric mucosa or from small erosions are dependent on portal congestion. A sharp purgative followed by tannigen or bismuth is sufficient to check the bleeding. But for hæmatemesis caused by dilated œsophageal veins the treatment must

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be the same as in cases of gastric ulcer. Absolute rest and rectal feeding for a week or ten days are the essentials.

When once *ascites* is established it is too late to do more than treat it by paracentesis abdominis. Death nearly always takes place after the first or second tapping. Earlier in the disease the onset of *ascites* may be delayed or prevented by encouraging the formation of an efficient collateral circulation between the portal and systemic veins. This can be done by the Talma-Morison operation, which consists in scraping the peritoneum of the liver and diaphragm so as to cause extensive adhesions. Sometimes it is further promoted by interposing the great omentum between the two raw surfaces. Epiploxy or suture of the omentum to the anterior abdominal wall is less severe but less effective.

Operation may prevent *ascites*, but is of still more value in encouraging hyperplasia of the liver-cells, because the degenerative changes in them are the cause of death in many cases. If performed at all, it ought to be undertaken early in the disease.

For *toxic symptoms* such as delirium the best treatment is intravenous salines or salines to which sodium bicarbonate (2 dr. to a pint) is added. As a rule, this is only a means of prolonging life for a short time.

BILIARY CIRRHOSIS (UNILOBULAR CIRRHOSIS)

Three forms of biliary cirrhosis are met with:

1. Hypertrophic biliary cirrhosis, or Hanot's cirrhosis.
2. Biliary cirrhosis of infants.
3. Obstructive biliary cirrhosis.

The first two will be considered together, the third separately.

Etiology.—*Hypertrophic* biliary cirrhosis is commonest between the ages of 20 and 30, but a number of cases occur in young children. Both sexes are equally affected. Several cases often occur in one family, and more than one generation may suffer. The disease is very rare in the British Isles, but is much commoner on the Continent. Alcohol, syphilis, and malaria play no part in its production. The typhoid group of diseases is thought by some to be the primary cause. The poison is conveyed by the hepatic artery and is not due to ascending cholangitis. In the later stages of the disease multilobular cirrhosis may develop.

Biliary cirrhosis of infants begins when the child is a few months old, and occurs chiefly

in Hindus in India. Pericellular and unilobular cirrhosis are both present. It is probably due to a specific infection. A form of biliary cirrhosis like Hanot's cirrhosis, but more rapidly fatal and without enlargement of the spleen, is found in Mexico.

Congenital stenosis of the bile-ducts probably begins as a biliary cirrhosis.

Pathology.—In Hanot's cirrhosis the liver is always very large with a smooth or finely granular surface. On section it is firm and dark green in colour. The meshwork of connective tissue is much finer and more uniform than in multilobular cirrhosis. The bile-ducts appear normal, and viscid bile is found in the gall-bladder. There may be local adhesions with subcapsular fibrosis. The liver-cells show little change until near the end of the disease, when degenerative changes begin. The connective tissue is much less dense and more open in arrangement than in portal cirrhosis. It is fairly uniformly distributed round each lobule, and some fine intracellular strands develop. Newly formed bile-ducts are generally numerous. The small bile-ducts show evidences of inflammation and may be obliterated, and fibrosis occurs round them. Small calculi are sometimes seen. The spleen is always very large, two to six times the normal weight, and is largest in children. The capsule is often adherent.

Symptomatology.—The chief characters of the disease are jaundice with periodic exacerbations, accompanied by fever and enlargement of the liver and spleen. The spleen is very often enlarged before any other indication of disease appears, and in the families affected some members never show any sign other than splenic enlargement. In other cases jaundice is the first sign, or loss of appetite, vomiting, diarrhoea, and hepatic pain. In the intervals between the exacerbations the tongue may be clean and the appetite good. The faeces are highly coloured and there is often diarrhoea. Polyuria is present. Bile-pigment and urobilin are found in the urine, which shows little tendency to deposit urates. During exacerbations there are often pain and tenderness over the liver and vomiting; the stools may become pale and the urine be diminished. The liver feels very large, firm, and smooth, and the large hard spleen is easily palpable. The abdomen appears very large, especially in its upper half. Nutrition remains comparatively good for a long time. When the disease starts in childhood, development is retarded and the fingers may become clubbed.

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Only in those cases in which portal cirrhosis develops and proves fatal is hæmatemesis or ascites met with. In the others death takes place from toxæmia or some intercurrent disease. The illness may last from five to ten years.

Diagnosis.—Some cases resemble the acute form of *portal cirrhosis*, but in this the duration is shorter and the jaundice transient. *Hæmochromatosis* is distinguished by the absence of true jaundice and the frequency of glycosuria. *Calculi* in the bile-ducts may produce intermittent fever and exacerbations of jaundice, but they occur late in life, produce greater pain, and cause much less splenic enlargement. In *hamolytic jaundice* the jaundice is lighter and there is no bile in the urine. The liver is smaller and there is fragility of the red corpuscles with anæmia. This disease runs a much slower course. *Syphilis* may present some clinical resemblances, but the Wassermann reaction is positive and other syphilitic lesions may be present. *Tuberculosis* in rare cases reproduces all the characters of Hanot's cirrhosis. *Splenic anæmia* may be confused with cases in which splenic enlargement is the only sign of the disease, but in splenic anæmia hæmorrhages and anæmia are likely to occur, whereas at this stage biliary cirrhosis gives rise to no symptoms.

Prognosis.—Infrequency of the febrile periods and maintenance of good nutrition indicate a prolonged lease of life. Rapid increase in the size of liver and spleen, with frequent attacks of fever and steady loss of weight, signifies that death will take place relatively soon. Signs of portal cirrhosis make the prognosis very bad. All cases end fatally.

Treatment.—No treatment can be more than palliative. A diet richer in fat and protein can be allowed than is permissible in portal cirrhosis. Salines are valuable. Intestinal antiseptics, such as beta-naphthol 5 gr. t.d.s., or calomel, have been recommended, and hexamine (urotropine) 5 gr. t.d.s. may help to check the cholangitis. Pruritus due to jaundice may be allayed by calcium lactate 10 gr. t.d.s. by the mouth, and by sponging the body with carbolie acid (1 in 40) at bedtime. Permanent drainage of the gall-bladder has given good results.

OBSTRUCTIVE BILIARY CIRRHOSIS

Etiology.—This is a rare sequel to obstruction of the bile-ducts, and is generally due to calculous obstruction. The modern tendency to operate early has diminished it still further.

It is not dependent on the obstruction itself, but on secondary infection of the larger bile-ducts. It is commoner in women than in men, and usually affects the middle-aged.

Pathology.—The liver is enlarged in the early stages, but by the time cirrhosis is established it is smaller than normal. On section it is much softer than the liver of multilobular or hypertrophic biliary cirrhosis, and numerous dilated bile-ducts are visible, chiefly near the surface. The surface is irregular and dark green in colour. Only the larger ducts are affected, and they show evidences of cholangitis. The cirrhosis is due to the spread of the infective process from the interior of the ducts, and so is chiefly a biliary cirrhosis of the larger portal spaces. By an extension of the inflammatory process a true multilobular cirrhosis may arise, and some intralobular cirrhosis may be visible under the microscope. The liver-cells become atrophic, and necrotic areas are generally present.

Symptomatology.—The symptoms of cirrhosis are quite secondary to those of biliary obstruction. The outstanding features are jaundice with pale stools due to the obstruction, and toxæmia due to the damaged liver-cells.

Diagnosis.—The deep and persistent jaundice and pale stools separate this condition from *portal cirrhosis*. The small size of the liver and of the spleen are important points in separating it from *hypertrophic biliary cirrhosis*. The stools are pale, or if a stone has been passed there is a history of pale stools having occurred earlier in the disease. The pain of gall-stone colic is much more severe than that of hypertrophic biliary cirrhosis.

Prognosis.—This depends on the extent of the cirrhosis and of the damage to the liver-cells. It can only be ascertained by watching the result of treatment, which in calculous obstruction consists in removing the stone or stones. When the jaundice has persisted for a long period, or when symptoms of toxæmia are prominent, the outlook is bad.

Treatment.—The only adequate treatment is to remove the obstruction, which is usually a calculus. Operation is useless when the calculus has caused complete obliteration of the lumen of the duct from cholangitis and in most non-calculous cases. After-treatment for the cirrhosis is the same as for portal cirrhosis, except that salicylates or hexamine (urotropine) should always be employed for the inflammation of the bile-ducts.

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LIVER ENLARGEMENT, DIFFERENTIAL DIAGNOSIS OF

LIVER ENLARGEMENT, DIFFERENTIAL DIAGNOSIS OF.

—True enlargement of the liver must be distinguished from *apparent enlargement*, such as that produced by displacement of the organ downwards or forwards (*see* VISCEROPTOSIS). Careful physical examination, supplemented if necessary by screen examination with X-rays, will generally distinguish the one from the other. Well-marked cases of wandering or floating liver are rare, but minor degrees of *hepatoptosis* are not uncommon. True enlargement of the liver may also be simulated by *congenital abnormalities*. In rare cases the left lobe of the liver is completely or almost completely absent, and the right lobe is correspondingly enlarged. The total amount of liver substance is not increased, but the right lobe forms a large abdominal tumour. Large or small pedunculated masses of liver of congenital origin arising from the lower border of the right lobe have also been described. A similar process known as Riedel's lobe may be caused by cholelithiasis or by tight lacing. As a rule, none of these abnormalities gives rise to symptoms, but they have been mistaken for renal tumours, pancreatic cysts, and other abdominal tumours. If they cause pressure symptoms or pain, they may be successfully removed. In the case of Riedel's lobe the use of looser corsets and treatment for gall-stones often relieve the symptoms. In some cases showing no symptoms laparotomy has been performed, and is the only sure means of diagnosis.

True enlargement of the liver takes the following forms:

NEW GROWTHS.—These may be simple or malignant, primary or secondary. *Simple* new growths are seldom large, and the large ones are only recognized by laparotomy. *Malignant* growths, usually secondary, are the commonest of all causes of enlargement of the liver. The organ is often painful and tender, with nodules of varying size on its surface. Rapid increase in the size of individual nodules or umbilication distinguishes this condition from syphilis or cirrhosis. Jaundice occurs in half the cases, ascites is less common. Pyrexia is often present. Rapid wasting of the patient is very characteristic.

HYDATID CYST.—Enlargement is usually local. The liver is smooth and not hard. Hydatids near the lower border feel tense and may give the "hydatid thrill," those on the upper surface often form a projection recog-

nizable by X-rays. Eosinophilia is sometimes present. The general condition is good in the absence of suppuration.

SYPHILIS.—The liver is very irregular. Splenic enlargement is common. Jaundice is rare, but ascites is commoner. The general health is well maintained, and local symptoms are slight or absent. The Wassermann reaction is positive, and other evidences of syphilis may be found.

TUBERCULOSIS.—The liver is seldom much enlarged. Jaundice is very rare. Other forms of abdominal tuberculosis almost invariably coexist.

ACTINOMYCOSIS.—Clinical recognition is unlikely unless the disease is known to occur elsewhere, or the chest or the abdominal wall is penetrated.

AMEBIASIS.—This may cause hepatitis or tropical abscess. There is usually a history of dysentery. Pain, swelling, and fever are present. Rigors are not uncommon, jaundice is very rare. Local swelling on the upper surface may be revealed by X-rays. An abscess in this situation may simulate empyema.

MALARIA.—The spleen is generally enlarged more than the liver. There are wasting, cachexia, and anæmia, with leucopenia. Fever is seldom high. Slight icterus may be present. There is often a history of malarial attacks, and the parasite may be present in the blood.

KALA-AZAR.—There may be great hepatic and splenic enlargement with low fever and anæmia. The disease is confined to the Mediterranean area and the East. Leishman-Donovan bodies may be found by splenic puncture.

LYMPHADENOMA.—The liver is smooth, hard, painless, and not tender. Jaundice is rare. The spleen and lymph-glands are generally enlarged. Fever is present and may be periodic.

SPLENIC ANÆMIA.—The spleen is much more enlarged than the liver. Early hæmatemesis is common. There is anæmia with leucopenia. The skin may have a faint yellow tinge.

HÆMOLYTIC JAUNDICE.—The liver and spleen are large and hard. There is slight permanent jaundice, with urobilinuria. The red blood-cells are unduly fragile and reduced in number. The general health is fair.

LYMPHATIC AND SPLENOMEDULLARY LEUKÆMIA.—Both diseases cause enlargement of the liver and spleen and anæmia. Diagnosis is rendered easy by means of a differential blood-count.

LIVER ENLARGEMENT

LIVER, NEW GROWTHS OF

PSEUDOLEUKÆMIA INFANTUM OF VON JAKSCH.—This is a disease of infancy and is generally accompanied by great splenic enlargement. Petechial hæmorrhages are common. There is marked lymphocytosis, and myelocytes and nucleated red cells are present.

PORTAL CIRRHOSIS OF THE LIVER.—The organ is hard, and uniformly covered with small or medium-sized nodules. Pain and tenderness are absent or slight. Enlargement of veins, where the systemic and portal circulations communicate, is common. Hæmatemesis and dyspepsia are frequent. An alcoholic history is usual. Ascites and slight jaundice often develop towards the end of the illness, and pyrexia, capillary oozing, and emaciation also occur at this stage.

HYPERTROPHIC BILIARY CIRRHOSIS.—The liver is large and feels smooth and firm, and the spleen is greatly enlarged. Jaundice is permanent and the stools are generally dark. The disease occurs only in young people.

HÆMOCHROMATOSIS.—Bronzing of the skin, enlargement of the spleen and glycosuria make established cases easy to recognize.

CONGENITAL STENOSIS OF THE BILE-DUCTS.—This condition is confined to babies. There is progressive jaundice with white stools. The spleen may be enlarged. Hæmorrhages are common.

BILIARY OBSTRUCTION.—The liver at first is large and smooth. There is progressive jaundice, and a history of biliary colic is often obtainable.

CHRONIC VENOUS CONGESTION.—The liver is tender and painful, and may pulsate. Pulsation in the veins of the neck may be seen. Albuminuria, œdema of the legs, cyanosis and ascites are often present. Signs of chronic cardiac or pulmonary disease are recognizable.

FATTY LIVER.—The organ is smooth and soft. Signs and symptoms of local disease are absent.

AMYLOID DISEASE.—This is found in syphilis, and also in long-continued suppuration, especially when associated with tuberculosis. The liver is smooth, very hard, and may be much enlarged. Ascites is common. Amyloid disease elsewhere may cause splenic enlargement, pronounced albuminuria, and diarrhœa.

PERIHEPATITIS.—The edge of the liver is never much below the costal margin, and feels smooth, hard, and thick. Ascites is very common, but jaundice is absent. For a long time the general health is good.

TOXÆMIC JAUNDICE.—There is usually a

prodromal period, and the duration of the disease is short. Jaundice and anæmia are present in varying degrees. Fever is common and toxæmia profound.

CATARREAL JAUNDICE.—The enlargement of the liver is slight. Jaundice is often deep, and associated with pale stools. Children and young adults are most often affected. The disease is usually brief and benign.

MULTIPLE ABSCESS OF THE LIVER AND PYLE-PHLEBITIS.—The liver is large and tender. Continuous fever is present, and there are often rigors. There may be some jaundice. The condition is obviously very serious from the beginning.

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LIVER, FATTY (see LIVER ENLARGEMENT, DIFFERENTIAL DIAGNOSIS OF).

LIVER, HYDATIDS OF (see HYDATID DISEASE).

LIVER, NEW GROWTHS OF.—Hepatic new growths may be innocent or malignant, and the malignant growth may be primary or secondary.

Etiology.—Innocent growths are common and are nearly always angiomas. They are usually small and single, but large encapsulated ones are found occasionally.

Primary malignant growths may be carcinomata, sarcomata, or endotheliomata; they are all rare. In adults the average age of incidence is over 40. Most of the cases in children described as primary sarcoma are syphilitic.

Secondary malignant growths in the liver are much more common, occurring twenty to forty times as frequently as primary. Carcinoma is about four times as common as sarcoma. The average age-incidence is about 50. In carcinoma the most frequent sites of the primary growth are the stomach, colon, œsophagus, and pancreas.

Pathology.—Primary carcinoma is single and massive, diffuse, or multiple, or it occurs in association with cirrhosis of the liver, producing cirrhosis carcinomatosa.

In **secondary malignant disease** the growths are nearly always multiple and vary in size. They may be white, bile-stained, or hæmorrhagic, and may be hard, or soft and even cystic owing to degenerative changes. Umbilication is common in the slower-growing nodules. The liver is increased in size, and its greatest known enlargement is met with in this disease.

LIVER, NEW GROWTHS OF

Symptomatology.—**Angiomata** seldom give rise to symptoms, but very large ones may cause pressure or hæmorrhage.

In **primary malignant disease** the symptoms differ little from those in extensive secondary new growths. Ascites is the rule in those associated with cirrhosis.

Secondary malignant disease causes no hepatic symptoms in half the cases. In the others, loss of weight and anæmia may be the first signs. Later the patient becomes very weak and wasted, with sunken eyes and sallow complexion, and with a peculiarly inelastic skin.

The abdomen may be greatly distended and covered with dilated veins. The liver rapidly enlarges, and nodules of various sizes, some umbilicated, can often be felt on its surface. Usually it is tender and painful. Jaundice supervenes in about half the cases, and progressively deepens. The gall-bladder is generally enlarged. Pruritus may be very troublesome. Ascites is a little less frequent than jaundice. Edema of the legs may be caused by pressure, heart failure, or toxæmia. Albuminuria is unusual. Fever is not at all uncommon and may be accompanied by rigors and leucocytosis. Secondary anæmia frequently occurs.

Diagnosis.—Large **angiomata** cannot be diagnosed without exploratory laparotomy. Most cases of **malignant disease** are easy to diagnose, others so difficult that laparotomy is necessary. In a doubtful case examination under an anæsthetic is often sufficient.

A **large cirrhotic liver** may simulate malignant disease, but the enlargement of the liver is more uniform and the nodules smaller and more numerous. The spleen is often enlarged and the cachexia is of slower development. When ascites is present, tapping may be necessary in order to facilitate examination. A **gummatous liver** is often very like one infiltrated by growth, but the pain is less, the increase in size much slower, and general health less impaired. A positive Wassermann reaction and improvement with iodides will settle all doubt. Simple and multiple **hydatid cysts** can be distinguished by their slower growth and smooth surface and by the absence of wasting. **Pylephlebitis** and **hepatic abscess** generally cause more fever, but may be simulated by a rapidly growing neoplasm. An **impacted calculus** in the common duct causes enlargement, but this tends to decrease, and the liver is smooth. The jaundice usually

LIVER, SYPHILIS OF

lessens after a time, whereas in malignant disease it deepens. In cholelithiasis the gall-bladder is contracted as a rule; in new growth it is distended. A **chronically congested liver** has been mistaken for malignant disease. The general appearance of the patient and his history, together with the physical signs of heart disease, ought to prevent this error.

In all cases the discovery of a primary growth, or of secondary deposits near the umbilicus or in the glands above the left clavicle, will remove all doubt.

Prognosis.—**Primary malignant disease** progresses rapidly, and usually causes death in two to four months. In **secondary malignant disease** death usually occurs within six months of hepatic enlargements.

Rapid increase in size of the liver, rapid wasting and symptoms of toxæmia indicate an early termination.

Treatment.—For large **angiomata** the treatment is excision. In **malignant disease** only palliative treatment is possible. For pain, aspirin, morphine, and opium compounds must be used, and the opium derivatives need not be restricted in a disease which lasts only a few months. Hot fomentations may give additional relief. For pruritus the best drugs are morphine or pilocarpine, gr. $\frac{1}{2}$, given subcutaneously, and calcium lactate by the mouth. Alkaline baths or sponging with carbolic acid (1 in 40) are also valuable. For vomiting, morphine, dilute hydrocyanic acid and bismuth, cerium oxalate and tincture of iodine may be tried. Flatulence may be controlled by creosote or menthol pills, and constipation is treated best by cascara or senna pods and salines. Ascites may require paracentesis abdominis.

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LIVER, PUNCTURE OF (see KALA-AZAR; TROPICAL ABSCESS).

LIVER, RUPTURE OF (see ABDOMINAL INJURIES).

LIVER, SYPHILIS OF.—Infection of the liver by the *Spironema pallidum* results in the following lesions:—

SECONDARY.

1. Pericellular cirrhosis.
2. Cholangitis of the small bile-ducts.
3. Fatty degeneration or necrosis of the liver cells.

TERTIARY.

1. Gummata.
2. Cirrhosis.

LIVER, SYPHILIS OF

Indirect effects of syphilis are multilobular cirrhosis and lardaceous disease.

Etiology.—**Infection before birth** occurs through the umbilical vein, and the disease is present at birth. When infection occurs during birth, the child appears healthy at first and shows signs of disease a month or six weeks later. It is commoner in female than male infants.

Congenital tertiary lesions develop in childhood, adolescence, or later. They may appear at any age between 10 and 30, but are commonest about puberty.

Acquired secondary syphilis chiefly affects young adults, especially males. **Acquired tertiary lesions** do not become manifest as a rule until late middle age, often twenty years after the primary infection.

Multilobular cirrhosis due to toxins of various kinds acting on a liver injured by syphilis may appear in childhood, but is much commoner in middle-aged adults. Universal lardaceous disease occurs late in the tertiary stage.

Pathology.—The liver in **early congenital syphilis** is enlarged, smooth, and firm. On section it cuts with more difficulty than usual, but may present no abnormal appearance to the naked eye. Microscopically there is seen to be diffuse uniform infiltration with round cells, which lie between the lobules, in the portal spaces and between the individual liver-cells. Later, newly formed connective-tissue cells can be seen in these positions, giving rise to a true pericellular cirrhosis. Sometimes the organ is studded with grey specks, which are seen to be minute syphilomata or gummata. By special staining, spirochaetes are seen throughout the liver.

In **secondary syphilis in adults** there are fatty degeneration of the liver-cells, which may proceed to necrosis, and cholangitis of the small bile-ducts, which causes jaundice. Pericellular cirrhosis is probably not very rare.

In **late hereditary and acquired syphilis** the liver presents very varied appearances. Syphilomata, appearing as pink masses of granulation tissue, constitute the first stage; these form gummata, which are visible as spherical round masses surrounded by pinkish connective tissue. Giant cells are often seen in them at this period.

Later, caseation takes place and the fibrous tissue increases and loses its pink tint. Some caseous gummata become softened and others calcified, but the majority become partially

or completely absorbed and the contracting fibrous tissue round them leaves deep scars. In addition, fibrosis takes place in the portal spaces. Syphilomata and gummata in all stages may be present at the same time, or the majority may be at the same stage of evolution. In hereditary cases there may be some pericellular cirrhosis also.

In the immediate neighbourhood of the gummata there are syphilitic endarteritis and lardaceous disease. Perihepatitis is found over superficial gummata. Gummata appear as spherical greyish or yellowish-white masses varying from the size of a pea to that of an orange. Most of them lie deep within the substance of the liver. When they have been numerous and the majority have been absorbed, the condition is known as syphilitic cirrhosis.

The liver is often contracted and extraordinarily distorted. Its capsule is locally or universally thickened, and nodules of all shapes and sizes project between deep fissures. The organ is very hard, and on section shows areas of normal liver intersected by bands of fibrous tissue. The liver-cells which are not compressed appear fairly normal on microscopic examination. The spleen is almost always large, hard, and fibrotic, and perisplenitis is generally present.

A gumma may press on a bile-duct, or fibrosis may constrict it, and so produce jaundice, but this is unusual. Ascites is a less uncommon sequel.

Symptomatology.—No symptoms attributable to the liver condition are produced in **infancy**. Jaundice is caused in **secondary syphilis in adults** by cholangitis. Necrosis of liver-cells with auto-intoxication may cause acute yellow atrophy, but this is very rare. In the **tertiary stage** perihepatitis may cause pain in the right hypochondrium. Pressure of a gumma in the portal fissure may produce jaundice with attacks of pain and ascites. In the early stage there is little interference with the general health, later there may be cachexia and anaemia.

Diagnosis.—In **infants** other manifestations of the disease are usually present, such as wasting, earthy complexion, rash, mucous patches, and anal condylomata. The liver is hard and smooth and may be felt below the umbilicus, the spleen is enlarged and hard. Jaundice and ascites are both rare. The mother will often give a history of previous miscarriages. The condition with which con-

fusion is most easy is pseudo-leukæmia infantum of von Jaksch, but a blood-count will diff. entiate the two conditions.

The subjects of **congenital tertiary syphilis** are often undersized and infantile and have a characteristic facies. Rhagades, interstitial keratitis, Hutchinson's teeth, and periostitis often accompany it, and the fingers may be clubbed. The liver is hard and irregular, and ascites is often met with owing to portal obstruction or peritonitis. The spleen is large and hard.

In **acquired secondary syphilis** implication of the liver is indicated by the development of jaundice. It is always associated with other signs of the disease such as sore throat, rash, mucous patches, and condylomata.

In **acquired tertiary syphilis** other manifestations are less often present than in congenital cases, but a history of a primary sore may be obtained. In *portal cirrhosis* there will often be found a history of dyspepsia and alcoholism, and the general health suffers more. Hæmatemesis and jaundice are much commoner in cirrhosis. When the liver is enlarged in syphilis its extreme irregularity makes diagnosis easy. In *malignant disease* the liver may be as irregular as in syphilis, but umbilication can generally be detected in some of the nodules. Rapid increase in size of individual nodules or of the whole liver is in favour of new growth, as are jaundice, pain, and tenderness. Enlargement of the spleen is in favour of syphilis. Rapid wasting is common in new growth. When the liver is enlarged, the smooth round edges in *perihepatitis* contrast with the nodular liver of syphilis, but when the organ is contracted, diagnosis may be very difficult. Ascites is commoner in perihepatitis. The chronic course, anæmia, and cachexia of severe visceral syphilis make it superficially resemble *splenic anæmia*, but hæmatemesis is much commoner in the latter disease.

In all forms of syphilis the Wassermann reaction is generally positive and may settle a doubtful diagnosis.

Prognosis.—When the disease has been contracted *in utero* the child is often stillborn, or dies soon after birth. When infection has occurred **during birth** the risk to life is considerable. The larger the liver, the worse is the prospect of recovery. Jaundice is unfavourable. **Acquired secondary syphilis** of the liver is usually benign, but prognosis must be guarded owing to the possibility of acute

yellow atrophy. In **congenital or acquired tertiary syphilis**, enlargement of the liver and jaundice or ascites due to gummata yield readily to treatment, but when the jaundice or ascites is due to cicatricial tissue and cirrhosis is established, the prognosis is bad, though life may be prolonged for years.

Anæmia, cachexia, and lardaceous disease are bad signs.

The results of treatment afford the only means of accurate prognosis.

Treatment.—In severe cases in **infancy** it is best to begin treatment by mercurial inunction, $\frac{1}{2}$ dr. daily, and later to give hydrarg. c. cret. $\frac{1}{2}$ gr. t.d.s. Diarrhœa can be controlled by adding pulv. ip. cæc. co. $\frac{1}{2}$ gr. to the grey powder. The arsenical compounds are still on trial. Good results have been obtained with sulpharsenol, which causes no local disturbance, and can be given subcutaneously or intramuscularly. Mercury should be used as well. Iodides should be given in the second and third year. This may produce a complete cure even in severe cases, and the Wassermann reaction may become negative. In **secondary and tertiary syphilis**, the best treatment is novarsenobillon or some other arsenobenzol compound, but the dose must be small, as these compounds are liver poisons and the liver is already damaged by syphilis. For an adult 0.3 grm. kharsivan is enough, and for a child the dose must be still less. The arsenic is not eliminated for three weeks at least, so the interval between the doses must be long. The urine ought to be examined for albumin and casts, and, if syphilitic nephritis or any other form of kidney disease be present, mercury and iodides are preferable to arsenical compounds.

Arsenical treatment must be supplemented by intramuscular infections of grey oil once a week, or by mercury and iodides by the mouth.

E. A. COCKAYNE.

LIVER, TUBERCULOSIS OF.—Tuberculosis of the liver occurs in four forms:

1. Miliary.
2. Massive.
3. Tuberculosis of the bile ducts.
4. Glandular.

Etiology.—**Miliary tuberculosis** either forms part of a generalized tuberculosis in which bacilli have been carried by the hepatic artery from an older tuberculous lesion, or is secondary to an abdominal focus from which the bacilli have been conveyed by the portal vein.

I have seen two cases in which no primary focus was found and the liver alone was involved. It is commonest in children.

Massive tuberculosis and tuberculosis of the bile-ducts are nearly always associated with other forms of abdominal tuberculosis. Both are rare, but are more frequent in children than in adults.

Tuberculosis of the lymphatic glands of the portal fissure is very rare, because these receive their lymph from the liver.

Pathology.—**Miliary tubercles** are found under the capsule and in the substance of the liver.

In **massive tuberculosis** a single or, more often, several large caseous masses, firm and white on section, are found in the liver. Sometimes they break down and form abscesses.

Tuberculosis of the bile-ducts begins as a miliary infection conveyed by the portal vein. Tubercles are produced in various parts of the portal system and, after several months, break through into neighbouring bile-ducts, and form cavities varying in size from a pea to a walnut, filled with green fluid. Local cholangitis occludes the duct above and below. The liver is moderately enlarged and no jaundice is produced.

Symptomatology.—**Miliary tuberculosis** produces no symptoms due to involvement of the liver. **Massive and biliary tuberculosis** cause no special effect but merely increase the severity of the general symptoms. **Glandular tuberculosis** may cause jaundice.

Diagnosis.—**Massive tuberculosis** cannot be diagnosed during life unless a caseous area be situated near the surface. If a lump be felt projecting from the surface of the liver in a case of abdominal tuberculosis, it will be due most probably to this condition.

Biliary tuberculosis is seldom diagnosed. Moderate enlargement of the liver in a case of protracted abdominal tuberculosis may be due to it, but is more usually due to fatty degeneration.

Prognosis.—This depends more upon the extent and character of the other tuberculous lesions than on the liver disease.

Treatment.—No special treatment is available. It must be directed to the primary abdominal lesion, usually tuberculous ulceration of the intestines or peritonitis.

E. A. COCKAYNE.

LOBULAR PNEUMONIA (*see* Bronchopneumonia, under PNEUMONIA).

LOCKJAW (*see* TETANUS).

LOCOMOTOR ATAXY (*see* TABES DORSALIS).

LORDOSIS (*see* SPINAL CURVATURE).

LUDWIG'S ANGINA.—A submaxillary cellulitis, usually due to a streptococcal infection, though staphylococci and pneumococci are also found. There is generally some primary disease in the mouth cavity or pharynx—a periodontal abscess, an infection of the sublingual or submaxillary salivary glands, a lingual cellulitis or abscess.

Symptoms.—A brawny area appears and spreads rapidly to the whole submaxillary region. The neck becomes stiff from the great swelling and infiltration which take place, and pain may be severe. The floor of the mouth is oedematous and perhaps brawny. Often the tongue is swollen, thrust forwards and partially immobilized; it is covered with fur. Tartar collects around the teeth and saliva dribbles from the mouth. The primary lesion may be found in the mouth. The constitutional symptoms are severe. The patient is ill and has usually been much depressed in health previously. The condition is serious because of its tendency to spread deeply and widely among the cellular fascial planes of the neck. Sudden oedema of the larynx is always to be borne in mind as a possible complication. Often there is dysphagia.

Treatment.—Owing to the danger of a sudden onset of acute dyspnoea it is best to make incisions early. Three are necessary: one in the middle line in front, and one on each side over the submaxillary triangle, parallel to the border of the lower jaw, the position of the facial artery being remembered. It is essential that these incisions should open up the deep fascia along their whole length if spread of the infection is to be stayed. As in other forms of cellulitis, no pus may be found, but the incisions are imperative. Boric acid fomentations are to be applied and the general condition strengthened. If it be possible to keep the part sufficiently immobilized by sandbags, Carrel's intermittent irrigation may be employed with advantage; but the patient is often ill and restless, and will not submit to this restraint. The tongue may require incisions, and an offending septic tooth may need extraction. C. A. PANNETT.

LOBAR PNEUMONIA (*see* PNEUMONIA).

LUETIN REACTION (see SEROLOGICAL DIAGNOSIS).

LUMBAGO.—Lumbago, or pain in the lumbar muscles due to pathological changes in them, is one of the commoner minor maladies. It occurs chiefly in middle and old age, and affects both sexes and persons in all stations of life.

Etiology.—Most cases of simple lumbago can be attributed to exposure to wet or cold, or to a chill; but the pain frequently starts after work has thrown an unaccustomed strain on the lumbar muscles, or after a strain due to injury or a violent contraction of the muscles. Its frequency in rheumatic subjects suggests that it is a form of muscular rheumatism or fibrositis; in certain cases it is probably due to other forms of chronic or subacute infection. Gout is in rare cases an etiological factor. Traumatic lumbago is due to physical injury of the tissues, as the result of a blow or a sudden wrench.

Pathology.—There have been few opportunities of studying the structural changes associated with lumbago; it is probably a form of fibrositis that involves the fibrous tissue within the muscles as well as their fascia, sheaths, and tendons. No evidences of inflammatory changes have been discovered, but an oedematous infiltration of the tissues is common. In traumatic lumbago various lesions have been found in muscles, particularly rupture or tearing of their fibres, small hæmorrhages, and a serous exudation, but the tendons and ligaments of the joints are frequently stretched or torn too.

Symptomatology.—The symptoms consist of pain referred to the lumbar muscles, and aggravated by movements that stretch them, and by rigidity of the lumbar spine. The pain often sets in suddenly after muscular effort, or after bending, or lifting a heavy weight. It is at first limited to the muscles, and is described as a deep-seated cramp-like pain; later it may become diffuse and spread over the ilium or into the loin. The pain may be absent during rest or in positions that allow the muscles to remain relaxed, but becomes severe when the muscles are stretched or called upon to contract by any movement. The spine is consequently held rigid, and the patient usually walks bent slightly forwards with short shuffling steps, as he avoids raising his feet by tilting the pelvis.

On examination the lumbar muscles are found to be tender; this can be demonstrated by merely pressing on them with the finger-tips

or, better, in lean subjects at least, by grasping them between the fingers inserted into the loin and the palm of the hand. The skin is rarely over-sensitive to touch or pinching, and pressure or percussion on the bones does not produce pain unless the ligaments and tendons inserted into them are involved. Stretching the muscles by forward flexing of the trunk, or by lateral flexion to the opposite side, also gives pain and may lead to a reflex or protective spasm of the muscles. Patients are usually most comfortable lying on the back on a firm bed.

Occasionally lumbago is associated with the symptoms of sciatica, owing perhaps to extension of the fibrotic changes to the sheath of the nerve, or to both conditions being due to the same cause.

Diagnosis.—Pain in the lumbar region is so frequently due to lumbago that other conditions which may produce it may by inadvertence be overlooked. *Disease of the lumbar and sacral spine*, especially caries or tumour of the vertebrae and spondylitis, must be carefully excluded. Caries and infiltrating tumours frequently lead to angular deformities, and they as well as spondylitis limit all movements of this portion of the vertebral column, not merely those that stretch the painful muscles. Further, these vertebral diseases develop more gradually and are more chronic than lumbago, in which the onset is usually sudden and the duration short. In *arthritis of the sacro-iliac joint* the pain is most intense over the joint and spreads over this half of the sacrum, which it fails to do in lumbago. *Intraspinous diseases* rarely produce symptoms that can be confused with lumbago, as pain excited by meningitis or by a tumour or other diseases of the posterior roots radiates round the whole side of the trunk and is not limited to the lumbar muscles. The lumbar pain of *renal disease* is more diffuse and widespread than that of lumbago, and is not associated with the same extreme tenderness of the muscles to pressure. The referred pains of *pelvic disease* and *colitis* may resemble those of lumbago, but they are more superficial, the skin is hyperæsthetic, they are scarcely influenced by movement, and the muscles are not so exquisitely tender to pressure.

Treatment.—Rest and warmth are the first two essentials; if the pain is at all severe the patient should be confined to bed, with hot applications to the lumbar region. Radiant heat is frequently very effective in relieving the more acute pain. When the pain diminishes in severity deep massage of the lumbar muscles

LUMBAR PUNCTURE

should be employed, combined with gentle active and passive movements of the back. The period of disability is certainly cut short by encouraging the patient to move about and bend his back when he can do so without great discomfort.

Various drugs, especially those that relieve rheumatism, as the salicylates, and particularly aceto-salicylic acid, are usually employed. They should be administered in large doses at frequent intervals during the first day or two. Iodide of potassium is of use in many cases. If the pain is very severe the coal-tar analgesics and gelsemium may be administered. As the condition may become chronic in inert and neurasthenic subjects, it is important to urge the necessity of movement and exercise when the severer pain has disappeared. Any chronic septic foci, as carious teeth, should be dealt with.

(GORDON HOLMES.)

LUMBAR ABSCESS (see SPINAL CARIES).

LUMBAR PLEXUS, LESIONS OF (see SPINAL NERVES, LESIONS OF).

LUMBAR PUNCTURE.—This operation is performed for the following reasons:

1. To remove cerebro-spinal fluid for diagnostic purposes and to estimate the pressure to which it is subjected. The procedure is always helpful and often necessary, both in the differential diagnosis of the varieties of meningitis and in their separation from polio-encephalitis and other simulating diseases. It assists also in the diagnosis of tabes dorsalis, general paralysis, and cerebro-spinal syphilis, by providing cerebro-spinal fluid for a Wassermann reaction.

2. To relieve pressure in cases of cerebral compression, especially in meningitis, in hydrocephalus, and in inoperable cerebral tumour. In meningitis its effect is perhaps more complex, for by the withdrawal of fluid the circulation is improved and more protective substances are brought to the inflamed meninges.

3. As a means of introducing medicaments. Examples are antimeningococcal serum in cerebro-spinal meningitis, antitoxin in tetanus, eucaïne in strychnine poisoning.

4. To produce "spinal anaesthesia" by the introduction of stovaine, novocain, or other local anaesthetic.

5. For the relief of uræmic coma or convulsions.

6. Excellent results have been recorded in cases of lichen planus.

Methods.—In the case of children who are conscious a general anaesthetic is necessary, but local anaesthesia may suffice for adults when the fluid is to be removed for diagnostic purposes. It is essential to obtain as wide a space as possible between the neural arches of the vertebrae. For this purpose, in the case of children the back should be arched by bringing the head into close proximity with the knees. When the patient's condition permits, the adult may sit up and bend forward with the head low. The needle selected should be 3 in. long, and preferably made of platinum-iridium. It should be sharp, but with not too fine a point. Some prefer a small trocar and cannula, but the needle has the advantage that it can be fitted to a syringe, by which suction may be employed if necessary. A sterile test-tube should be at hand to receive the fluid removed. The thecal space should be entered between the 3rd and 4th or 4th and 5th lumbar vertebrae. As a guiding line, that drawn between the highest points of the iliac crests is selected; this crosses the tip of the 4th lumbar spine, and the puncture may therefore be made either just above or just below this line. In the case of adults it is easier to insert the needle about half an inch from the mid-line, but in children the mid-line itself, or just to its side, provides readier access. The needle should be directed upwards and inwards, and its entry into the thecal space is readily appreciated by a sudden diminution in resistance; if bone is encountered the needle should be partly withdrawn and the direction of the thrust corrected. Usually the fluid will flow drop by drop through the needle, and should be collected in the sterilized test-tube. The first few drops may be allowed to escape, for they are not infrequently bloodstained. For diagnosis only 3 or 4 c.c. need be collected, but for therapeutic purposes more should be removed. Thus, for cerebro-spinal meningitis the amount of serum injected is 20-30 or 40 c.c., and more than this amount of fluid should have previously been withdrawn. The blood-pressure should be taken during the injection; a fall of 20 mm. Hg shows that the maximum amount has been given. (See also under IMMUNITY.)

The risks of infection are very small if proper precautions are taken, and no untoward symptoms follow provided that the fluid is not removed too quickly or in too great a quantity.

FREDERICK LANGMEAD.

LUNG, ABSCESS OF

LUNACY, CERTIFICATION OF (see INSANITY, CERTIFICATION OF).

LUNG, ABSCESS OF.—The term abscess of the lung is applied to any localized collection of pus, and to certain diffuse suppurative processes in the lungs, although it is usual to exclude softening tuberculous foci and dilated bronchi filled with pus.

Etiology.—The exciting cause is the presence of pyogenic organisms conveyed by inhalation, by the blood-stream directly or through infective emboli, or by direct extension from suppurative process in adjacent organs or tissues. The bacteria most commonly found are streptococci, staphylococci, bacillus coli, the pneumococcus and pneumobacillus, together with saprophytic or putrefactive organisms. Certain predisposing conditions are usually present promoting their action, and these include lowering of resistance of the lung due to disease, toxic agents or injury, and general debilitating conditions, notably diabetes.

The following comprise the chief conditions in which pulmonary abscess occurs:—

1. As a sequel of pneumonic processes, either lobar or lobular, especially the inhalation or aspiration forms induced by septic conditions about the mouth, naso-pharynx and upper air-passages.
2. Embolic abscesses from right-sided infective endocarditis, or from distant septic processes such as otitis, appendicitis, and thrombo-phlebitis.
3. Abscesses from extension, such as occur in association with bronchiectasis, carcinoma of the lung or œsophagus, growth or abscess in the mediastinum, caries of the spine, and abscess of the liver.
4. Following injury, including fractured ribs, perforating wounds, and inhaled foreign bodies.
5. Suppuration of a hydatid cyst before or after rupture, though this is not strictly an abscess of the lung.

Pathology.—Pulmonary abscesses may be single or multiple, localized or diffuse, acute or chronic. In cases following pneumonia, the abscess is usually basie, and often irregular in outline. The softened area is enclosed by œdematous and infiltrated lung tissue or by pneumonic consolidation. The close proximity of the bronchi renders early spontaneous rupture and evacuation common, so that a

definite fibrous wall is unusual, though this may develop around a chronic abscess, either before or after rupture.

Abscesses of embolic origin are generally small and multiple, and may develop in any part of the lung. Suppuration due to a foreign body or to extension often causes large irregular collections, involving a considerable area of a lobe. A superficial abscess induces pleurisy, sometimes proceeding to secondary empyema and to pyo-pneumothorax if rupture occurs.

Symptomatology.—Owing to the fact that abscess-formation is invariably a secondary process, the clinical manifestations are generally an aggravation of existing symptoms. The patient appears gravely ill and develops severe fever of remittent or intermittent type, not infrequently associated with rigors. Before rupture, the actual pulmonary symptoms may be represented only by cough with very little muco-purulent expectoration, some dyspœnea and pain, the latter sometimes due to pleurisy. At this stage a blood-examination may reveal a considerable leucocytosis. The sudden expectoration of a considerable quantity of pus, due to rupture into a bronchus, is often the first symptom suggesting abscess-formation. This is generally followed by a fall of temperature and an amelioration of the symptoms, except that cough is now worse. The sputum consists of pus with some debris of lung tissue, especially elastic fibres, often with an alveolar arrangement. It usually has an unpleasant musty odour, occasionally foul, but less so than in gangrene. If free drainage is established by rupture, the patient may recover rapidly. On the other hand, the pus may re-collect, the fever and other symptoms return, and the condition become chronic or progressive.

Physical signs.—A small deep-seated abscess gives rise at most to some local impairment of percussion note with weak breath-sounds and a few bubbling or crackling râles in the œdematous or consolidated lung tissue around. In embolic cases with multiple abscesses the signs may be indistinguishable from those of disseminated broncho-pneumonia. In localized abscess before rupture, they may simulate those of consolidation or encysted empyema. After rupture, signs of excavation are usually present.

X-ray examination is sometimes of great value, showing an opaque area before rupture and definite excavation afterwards.

Diagnosis.—Sudden copious expectoration

LUNG, ABSCESS OF

of pus containing elastic tissue, but no tubercle bacilli, after severe symptoms of septic type, is very suggestive of abscess. Before rupture, diagnosis is usually difficult. The condition may be suspected and exploratory puncture carried out, but in this case it is well to be prepared for immediate operation.

After rupture, the following conditions have to be distinguished: (a) Excavation in pulmonary tuberculosis. The history and the presence of tubercle bacilli in the sputum usually make the diagnosis clear. (b) Localized empyema, especially if of interlobar origin. (c) Bronchiectasis. (d) Gangrene of the lung. (e) Subphrenic abscess. (f) Fœtid bronchitis. The distinction from these conditions (except the first) is considered in LUNG, GANGRENE OF.

In embolic cases diagnosis may be almost impossible, the local signs being overshadowed by the general pyæmia.

Prognosis.—Apart from the pyæmic forms, the outlook, though grave, is not unfavourable. A fair proportion of cases in which spontaneous rupture occurs recover completely, and the chance of recovery is notably increased by operation in suitable cases. Cerebral abscess may develop as a complication.

Treatment.—When the abscess can be definitely localized before rupture, surgical treatment should be advised if the patient's condition will permit. When rupture has occurred the decision is more difficult. If the temperature remains unsteady and symptoms persist, operation is indicated, such as is described for gangrene of the lung.

If secondary empyema occurs, operation is imperative. For a large chronic cavity, some form of thoracoplasty may be necessary to allow the lung to shrink.

Medical treatment consists in sustaining the patient's strength, the evacuation of the cavity by postural manœuvres, and the administration of expectorants. Creosote, garlic, and the benzoates may be given if the expectoration is fœtid, and antiseptic inhalation such as creosote, carbolic acid, eucalyptol, and terebene should be employed, alone or in combination. Intratracheal injections of menthol 10 parts, guaiacol 2 parts, and olive oil 88 parts may also be helpful. The use of autogenous vaccines has seemed to be of real assistance in these cases, but should be avoided if drainage is unsatisfactory.

R. A. YOUNG.

LUNG, ACTINOMYCOSIS OF (see ACTINOMYCOSIS).

LUNG, COLLAPSE OF

LUNG, ACUTE SUFFOCATIVE CATARRH OF (see SUFFOCATIVE CATARRH, ACUTE).

LUNG, ATELECTASIS OF (see LUNG, COLLAPSE OF; and ATELECTASIS, CONGENITAL).

LUNG, CIRRHOSIS OF (see LUNG, FIBROSIS OF).

LUNG, COLLAPSE OF.—This term is indefinite and covers a large number of conditions. It is generally agreed that obstruction of a bronchus or bronchiole causes a collapse or deflation in the area supplied by it. Collapse, therefore, is present in bronchopneumonia in small areas, and in larger areas in such conditions as diphtheria producing a membrane in the bronchus, a foreign body inhaled and causing tumidity of the bronchial mucous membrane at the point of impaction, or a neoplasm obstructing the bronchus. The lung becomes deflated from pressure by surrounding material, which may be a serous effusion, pus, or a neoplasm filling up the pleural cavity. Deflation also occurs when the expanding force of the inspiratory muscular contraction is in abeyance.

In this article the last variety only is described; the remainder are discussed under the maladies with which they are associated.

Etiology.—If the supine position is retained for a period of 24–36 hours or longer, and the patient remains quiescent, the lower lobes of the lung become deflated, respiration being carried on by the upper and middle lobes. This is explicable as a result of diminished action of that part of the diaphragm that arises from the costal wall. The upper lobe of the lung is rarely deflated; when deflation occurs, it is due to paralysis of the spinal portion of the diaphragm or of the chest-wall in the upper part of the thorax. Deflation of the lower lobe is common after operations or such serious conditions as lead to complete quiescence in the posture indicated, and may be found in pronounced toxic fevers such as diphtheria, typhoid, etc.

Pathology.—Deflation may almost be regarded as normal, but becomes pathological when the deflated lung undergoes infection. This may occur when the patient operated upon is at the time suffering from a cold, bronchitis, etc., or when infected material is inhaled during anæsthesia. In an inactive state the lung is a favourable seat for the development of infection, which is liable to

LUNG, COLLAPSE OF

spread, and may be followed by consolidation of the finer parts of the lobe and pleurisy. Pleurisy may also arise from infection involving the parietal pleura covering the chest-wall or the diaphragm, and, in a patient with the lower lobes deflated, causes very serious symptoms.

Symptomatology.—Provided that there is no infection, there are few symptoms. A certain amount of shortness of breath may be noticed, especially when the patient begins to talk. It is then seen that breathing is quickened, only a few words being produced without the intervention of a respiratory effort. A frequent and non-productive cough, loose in character, may be present, and occasionally complaints are made of constriction about the upper part of the chest. When a mild bronchitis supervenes, the cough is similar but more troublesome, and great difficulty is experienced in expectoration. A rise in temperature often occurs; it is out of proportion to the physical signs, of which few can be detected.

Should pleurisy develop, its onset may be associated with very alarming symptoms. The patient sits up, struggles for breath, and becomes cyanosed; the respirations may rise to 50 or 60 per minute; the pulse becomes irregular, rising to 150 or higher; and pain is complained of in the region of the sternum or of the costal margins. This combination of signs has been termed a "collapse attack."

On careful examination an area of diminished resonance can be found posteriorly in the triangle formed by the spines of the vertebræ, a line drawn horizontally from the spine of the eighth or ninth vertebra and the fifth or sixth rib. Over this area the breath-sounds are diminished, indistinct, or bronchial; the voice-sounds bronchophonic or ægophonic. With a "collapse attack" there are signs indicating that the lung on the unaffected side is extremely hyperactive. On the affected side there is general deficiency of breath-sounds with lack of movement, especially noticeable in the upper part. Some râles are heard over the lower lobe, and examination of the heart will suggest that it is displaced towards the affected side. In the most serious cases pain is complained of in the shoulder as well as in the sternal region, and there is dysphagia. The temperature runs up to 103° F. or more within the next few days, and within this period, in most cases, friction is audible in the neighbourhood of the sixth rib or below.

Diagnosis.—When no symptoms occur, the condition will not be diagnosed unless a special examination is made. When the symptoms suggest *bronchitis* or *broncho-pneumonia* the signs of these maladies may be present, but in a much less degree than would be anticipated.

The "collapse attack" is to be diagnosed from pneumonia and from embolism. The onset of *pneumonia* is seldom so acute and is associated with rigor or vomiting; alarming symptoms do not ensue in the course of a few minutes. In pneumonia, again, the abdominal muscles on the side affected are tense; in the condition here described they are *lax*. *Pulmonary embolism* is equally sudden at its onset. When in embolism the patient's condition is so serious as to resemble a collapse attack, it is almost certain to be fatal, or, if not fatal, is followed by hæmoptysis considerable in degree.

Prognosis.—In ordinary cases of deflation following posture, recovery results, for directly the patient begins to move about and to breathe deeply the lobe becomes active. The collapse attack, although so alarming in character, practically always gets well, a considerable degree of relief ensuing within forty-eight hours. It may, however, be followed by pneumonia, bronchitis, etc., which will run their usual course.

Treatment.—Deflation, being a natural event, requires no special treatment. If a cough develops, it is advisable to make the patient expand his lower lobes by turning him to one side or the other and by making him talk and generally help himself. Such movements require the action of the abdominal muscles, which brings with it expansion of the lower lobes and should be practised. When the exigencies of the operation allow, patients should be raised on pillows on the following day at an angle of 40° or 60°. With bronchial infection, the usual treatment for bronchitis may be instituted. In the event of a collapse attack, nothing should be done to restrict the movements of the patient, who must be allowed to select that position which is most comfortable for him; hot applications to the painful areas are comforting, stimulants should be used if required, and an injection of morphia $\frac{1}{4}$ gr. is of great value. The subsequent course of the disease should be treated according to the indications.

In a few cases the lower lobes remain un-

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expanded; anything which will induce deep breathing such as a blow bottle, wind instruments, singing, and so forth, should then be tried. The patient should also be instructed to distend the abdomen—"abdominal breathing." If all these fail, the Japanese method of inserting a needle and exploring the lower part of the chest will almost certainly be effective. In order to achieve the maximum effect a local anæsthetic should be dispensed with.

Too much stress cannot be laid on the importance of preventing infection of the lungs during anæsthesia. Attention should therefore be given to the upper air-passages, the tonsils, teeth, and so forth. Operations subsequently involving the supine posture should not be undertaken, when they can be avoided, in the case of a patient with catarrh of the air-passages.

CHARLTON BRISCOE.

LUNG, CONGESTION OF.—The capillary vessels of the alveolar walls are dilated and filled with blood in a variety of conditions, both inflammatory and mechanical, grouped as congestion of the lungs.

The **inflammatory causes** of congestion may be divided into acute, as seen characteristically in the earliest stage of pneumonia, and chronic, as in pulmonary tuberculosis. The chief clinical interest of congestion is to indicate the stage of the disease which is present, and it should therefore be regarded more as an evidence of disease than as a clinical condition. It is present at the onset of pneumonia before consolidation takes place. The physical signs reveal little that is abnormal apart from a change in the percussion note and a diminished air entry. Congestion of the lung may also be encountered at the onset of typhoid fever. The early development and the peripheral extension of pulmonary tuberculosis are accompanied by local congestion.

The **pathological condition** shows in microscopical sections the alveolar walls distended and consisting almost completely of dilated capillaries with their contained red blood-corpuscles. Some exudates may be present in the alveolar spaces, with a few red corpuscles and epithelial cells in the fibrinous exudate where the inflammatory process is passing into the stage of consolidation. In tuberculous processes the characteristic multinucleated giant cell will be present with its surrounding cell-proliferation free from blood-

vessels, and congestion of the alveolar walls in the zone beyond this.

The **mechanical causes** of congestion are seen characteristically in cases of heart failure and, more particularly, in mitral stenosis. When the force of systole of the left auricle in conjunction with the suction action of the left ventricle is insufficient to overcome the obstruction to the flow of blood through the stenosed mitral valve, the auricle fails to empty itself. Blood is still being forced by the right ventricle into the alveolar capillaries, which dilate and become congested. This is chiefly seen at the bases, where gravity acts as an additional factor. Breathlessness develops and physical examination reveals dullness at the bases, diminished air entry, and crepitations, more obvious on the side on which the patient has been lying. This condition is present in many forms of heart failure, especially in disease of the mitral valve and the later stages of auricular fibrillation, and in auricular flutter. Other mechanical causes of congestion are mediastinal growths and thoracic aneurysms, while occupational kyphosis and skeletal deformities involving the chest also produce it. Finally, it is present in the later stages of severe infections, such as typhoid fever. It is almost consistently present in greater or less degree as a terminal condition in all except sudden deaths.

The **pathological condition** of congestion from mechanical causes shows the same dilatation of the capillary vessels, and a number of red blood-corpuscles in the alveolar spaces. These become foreign bodies; they are taken up by the epithelial cells and disintegrated, and the iron of the hæmoglobin is deposited in the lymphatics and alveolar walls, giving the characteristic appearances of "brown induration" of the lung.

A **diagnosis** of congestion of the lungs is no more final than would be a diagnosis of hæmoptysis. The essential diagnosis is that of the disease of which the congestion is only an accompaniment. The **prognosis and treatment** depend on the cause.

A. HOPE GOSSE.

LUNG, EMBOLISM OF.—A cutting off of the arterial blood supply to a portion of a lung as a result of obstruction to the lumen of a branch of the pulmonary artery by undissolved material conveyed in the bloodstream.

If the patient survives after the main blood

supply to a lobe or part of a lobe is cut off, and the collateral circulation is inadequate, an infarct of the lung results.

Etiology.—Embolism may result from the formation of a thrombus in the right side of the heart or in the pulmonary artery, associated with heart failure. A part of this thrombus becomes detached and obstructs the lumen of an artery supplying the lung.

Embolism may also occur as a result of thrombosis in the systemic veins and the detachment of a portion of the thrombus. Examples of this may be observed in such conditions of thrombosed veins as occur with varicose veins, in the convalescent stage of enteric fever, after parturition, in all cachectic and anæmic conditions, and following abdominal operations, especially appendicitis.

A rare cause is blocking of the terminal arterioles and capillaries by globules of fat after fractures of the long bones. These may be few and unaccompanied by symptoms and physical signs, or so numerous as to be the cause of sudden death.

Air-embolism, in which the capillaries are blocked by bubbles of air, may occur as a result of injury to a large vein in the neck and suction of air into its lumen. The small amount of air which is sometimes introduced into the veins in the administration of drugs by intravenous injections is not productive of symptoms, though naturally its admission should be avoided.

Symptomatology.—Sudden death results in those cases in which the blood supply to the whole or a large portion of one lung is cut off either by an embolism of a primary division of the pulmonary artery, as occurs in association with venous thrombosis, or by multiple emboli blocking numerous arterioles, as in fat-embolism.

When an artery supplying a smaller area of the lung has become occluded an infarct may be formed, the symptoms of which are of sudden onset and characterized chiefly by dyspnoea, hemoptysis (which may be profuse), and pain in the chest. The physical signs of an infarct are dullness over the infarcted area, bronchial breathing, increased vocal resonance and fremitus, and râles. Later a pleural rub may be heard over the pleural surface of the infarcted area, and this may go on to effusion.

As infarcts of the lung are often associated with heart failure and are a terminal event, the physical signs of an infarct are often obscured by those of passive congestion.

Embolism of the lung may occur without giving rise to any symptoms or physical signs. This is due partly to smaller arteries being affected, and more especially to the establishment of a satisfactory collateral circulation, and is dependent upon efficient action of the heart. An arteriole may be blocked without any death of the lung-tissue resulting.

Pathology.—The only essential feature in the pathology of embolism of the lungs is the presence in the blood-stream of undissolved material blocking a branch of the pulmonary artery. With infarcts of the lung there is necrosis of portions of the lung-tissue. Infarcts occur more frequently in the lower lobes than in the upper, at their bases rather than at their apices, more often in the right lung than in the left, and are more frequently multiple than single.

Diagnosis.—The presence of one of the diseases which give rise to embolism and infarcts, the sudden onset of symptoms referred to the lungs, and the development of the physical signs make the diagnosis probable. From pneumonia, embolism is distinguished by the lower temperature and the course.

Prognosis depends on two factors—the amount of lung-tissue which has been deprived of its circulation of blood, and the cause of the infarct. When sudden death has been escaped there is a tendency for pneumonia to develop. Apart from these considerations, the prognosis depends upon the cause.

Treatment is largely prophylactic, for embolism may often be avoided by careful nursing and adequate rest. The same measures are essential when embolism has taken place. Treatment must also be directed to removing the cause, since embolism and infarction of the lung are always secondary.

A. HOPE GOSSE.

LUNG, EMPHYSEMA OF (see EMPHYSEMA).

LUNG, FIBROSIS OF (*syn.* Chronic Interstitial Pneumonia).—A disorder of the lungs in which the predominant feature is a chronic inflammatory invasion of the alveolar or bronchial walls, with resulting scar-formation; it may either be diffuse or extend from the interlobular septa or the pleura.

Etiology.—The involvement of the tissues by fibroid changes usually begins insidiously, and is chronic from the outset, but it may be the result of an acute infection such as pneu-

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monia. It is seen at all ages, in children as commonly as in adults. In a rare form it is the result of a lobar pneumonia in which the exudate into the alveolar spaces has become organized and combined with fibrotic changes in the alveolar walls. It may result from a broncho-pneumonia, and sometimes appears to extend from the pleura as a result of acute pleurisy. Certain occupations, such as those of flint-knappers and stone-masons, involving the frequent inhalation of minute particles of silica which become lodged in the alveolar walls, produce a chronic inflammatory reaction. In many cases the cause cannot be determined, and it may be that a healed tuberculous focus, compression of a bronchus, or even syphilis is the hidden cause.

Symptomatology.—In a well-developed case the cardinal symptoms of diseases of the chest are present—shortness of breath, pain, and cough. The shortness of breath is noticed particularly on exertion, such as walking up hills or ascending stairs. At other times the breathing may be unaffected. Cough is very frequently troublesome and persistent; hæmoptysis occurs in many cases. For these reasons the condition is very frequently mistaken for chronic pulmonary tuberculosis. Pain in the chest may be present, but it is not a constant symptom. Clubbing of the fingers and toes is common, as also is cyanosis. The character of the sputum varies: it is frequently offensive when there is an accompanying dilatation of the bronchi or bronchioles. All symptoms are aggravated by an attack of bronchitis.

The **physical signs** are characteristic when the fibrosis is unilateral and diffuse. On inspection there is flattening of the chest, with hollows above and below the clavicle on the affected side. The shoulder is drooping, the intercostal spaces are reduced, and the spine is curved. The chest on the healthy side appears bulging in contrast. On palpation there is little or no expansion on the affected side, movement on inspiration being confined to the unaffected side. The vocal resonance is increased unless the fibrosis has resulted from an acute pleurisy with a consequent thickening of the pleura. The apex beat is displaced towards the affected side. The percussion note is dull over the whole of the fibrosed lung, whereas over the healthy lung the note may be hyper-resonant. The air entry is poor, and the breathing bronchial in character. Often coarse râles are heard, but at times the chest may be dry and moist sounds absent. In the opposite

lung the signs may be normal, or merely those of emphysema.

When the condition is bilateral the signs are very different, as both lungs share in the compensatory emphysema, which may partly or entirely mask the signs of fibrosis. Clubbing of the fingers may then help, as it is more constant in longstanding fibrosis than in any other condition; it serves to indicate to some degree the extent of the fibrosis when the heart is normal. Between the unilateral and the bilateral types of cases all degrees and grades may be found. For instance, the fibrosis may be more localized than in the unilateral type described, in which case it usually occurs at the base of the lung or near the hilum, though if it is the result of a healed tuberculous focus it will probably be near the apex.

Fibrosis accompanies many morbid conditions of the lung, such as chronic tuberculosis, bronchiectasis, abscess, hydatid, syphilis, and even chronic bronchitis and emphysema, but it is not the predominant feature in these cases, and so they have been excluded from consideration here.

Diagnosis.—In typical cases the diagnosis is simple and the physical signs are conclusive. But in very many cases of fibrosis a diagnosis of *chronic pulmonary tuberculosis* is made, and is sometimes only corrected by repeated examinations of the sputum for tubercle bacilli over a period of weeks or even months. This differentiation is of the utmost importance to the patient, as the presence of fibrosis is not inconsistent with a restricted life for very many years, whereas if the signs were attributed to active pulmonary tuberculosis the prognosis would be grave. In attempting to ascertain the cause in obscure cases, a Wassermann test may assist.

Treatment.—During the early stages of the disease resulting from unresolved pneumonia or pleurisy something may be done to minimize the damage. Painting the chest with iodine once daily for several days, combined with a prolonged course of full doses of potassium iodide, will help. I have known the moist sounds which at one time were very numerous entirely disappear during a course of creosote vapour inhalations for twenty minutes daily in a closed room. Motor goggles should be worn to protect the eyes from the fumes. In the later stages nothing can be done except the avoidance of the cause if still acting, symptomatic treatment, and the treatment of complications such as bronchitis and pneumonia as they

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arise. A carefully regulated sedentary life in a mild climate, when practicable, is not inconsistent with many years of comfortable life. Unless the patient succumbs to some acute complication or to hæmoptysis, eventual heart failure will probably bring about the termination.

A. HOPE GOSSE.

LUNG, GANGRENE OF.—Necrosis of localized or diffuse areas of lung tissue and their invasion by putrefactive organisms.

Etiology.—The disease is invariably secondary, and the necrosis of the lung tissue is usually induced by infection of areas in which resistance has been lowered by disease, toxic agents, or injury. It is favoured by debilitating conditions such as diabetes, alcoholism, and malnutrition, and is also commoner in the insane. The chief conditions leading to gangrene of the lung are—

1. Pneumonia, though gangrene is less common than abscess as a sequel of this disease.
2. Broncho-pneumonic processes, especially those due to the inhalation of septic particles or infective organisms from diseases of the mouth, naso-pharynx, larynx, and bronchi, or during anæsthesia. Among these diseases may be mentioned carcinoma of the tongue, mouth, or larynx, and laryngeal paralysis.
3. Foreign bodies inhaled into the air-passages.
4. Bronchiectasis; this not infrequently leads to gangrene, while tuberculosis only rarely does so.
5. Injury to the lung by gunshot or other wounds.
6. Invasion of the lung by growth originating in the œsophagus, the mediastinum, or the lung itself.
7. Obstruction to the blood supply by embolism or by pressure due to aneurysm or growth.

Pathology.—The organisms found are generally similar to those in abscess-formation, and include streptococci, staphylococci, pneumococci and pneumostreptococci, *Bacillus coli*, and putrefactive organisms such as *Bacillus proteus* and *Micrococcus tetragenus*. Acid-fast bacilli and anaerobes have also been described.

In circumscribed gangrene there is some attempt at forming a zone of demarcation,

consisting of congested lung tissue, while in the diffuse variety the process is progressive and unchecked. In either case the gangrenous area is soft and pulpy, while its colour is dark red-brown, green, or almost black, and its odour horribly fœtid. Softening occurs and putrefying necrotic masses may be found in a cavity containing purulent fluid. The surrounding lung tissue may be congested, œdematous or pneumonic. Ulceration of vessels may occur leading to hæmorrhage. The pleura over the necrotic area is generally acutely inflamed, and empyema or pyo-pneumothorax may result. When the necrotic débris has been expectorated, an irregular cavity is left with rough shaggy walls exuding offensive pus.

Symptomatology.—Invariably the patient is extremely ill. The symptoms are usually an aggravation of those previously present if gangrene supervenes on some pulmonary or intrathoracic disease. There is fever often of remittent or intermittent type. Rigors may occur at the onset or be repeated. The pulse is rapid, and profound prostration often develops early with restlessness and low muttering delirium. Cough is frequent and distressing. The sputum and the patient's breath are indescribably fœtid and the odour may pervade the whole room. The expectorated matter frequently amounts to several ounces daily. It varies in colour, being brown, greenish-yellow, or bloodstained. It separates on standing into three layers—a superficial frothy scum, a deep layer of brownish or green colour consisting of pus and necrotic tissue rich in micro-organisms, and an intermediate layer of turbid fluid. It invariably contains elastic tissue. Hæmoptysis is not infrequent both at the onset and during the course of the disease; it may be profuse and fatal.

Occasionally gangrene occurs without fœtid sputum, chiefly in diabetics, in the insane, and in young children, and also in cases due to embolism.

Physical signs.—These depend upon the stage, the situation, and the extent. They are not characteristic. In the early stages there are usually signs of consolidation. When softening occurs and the gangrenous tissue is expectorated, indications of excavation develop. As the case proceeds, signs of congestion, pneumonia, or œdema may become apparent in the unaffected part of the lung and in the sound lung. The signs of any pre-existing disease present, such as bronchiectasis, malignant dis-

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ease, or aneurysm, may complicate the clinical picture. If practicable, X-ray examination is often of great value in localizing the site of the process and in demonstrating cavity-formation.

Diagnosis.—The history, the grave condition of the patient, and the development under observation of signs of excavation, together with the characteristic fetid sputum, may render the diagnosis almost certain, but other affections causing fetid sputum should be eliminated. The most important of these are :—

(a) *Pulmonary abscess.*—The distinction between abscess and gangrene is not a sharp one. By some authorities all acute destructive processes in the lung associated with fetid sputum are classed as gangrene. In abscess there is generally a sudden and copious expectoration of pus which is less fetid than in gangrene, and is followed by amelioration of the symptoms.

(b) *Bronchiectasis.*—The history, the less acute progress, and the variable and rather characteristic physical signs usually suffice. In bronchiectasis, elastic tissue is absent from the sputum unless there are ulceration of the wall of the bronchus and destruction of lung tissue.

(c) *Fetid empyema*, particularly when it is interlobar in situation, may give rise to great difficulty. Signs of pleurisy early in the course, and evidence of mediastinal displacement, may assist, but both are not infrequently absent in interlobar empyema. The absence of elastic tissue from the sputum is a diagnostic point of importance.

(d) *Rupture of hepatic or subphrenic abscess through the diaphragm into the lung.*—The history and the evidence of disease or displacement of abdominal organs will generally suggest the origin of the pus, and elastic tissue will be absent from the sputum.

(e) *Fetid or suppurative bronchitis.*—There is a longer history, the sputum is less fetid, may occur only during bronchitic attacks, and does not contain elastic tissue.

Prognosis is always extremely grave, though a certain number of cases recover. The percentage of recoveries seems to be increased by operation in judiciously selected cases. Cerebral abscess and meningitis not infrequently occur as complications, and the liability to these is not lessened but rather increased by operation.

Treatment.—If the gangrenous area can be

localized by the physical signs and by X-ray examination, and if the general condition of the patient will permit, **operation** affords the best chance of successful treatment. Exploratory puncture is inadvisable, unless preparations for operation are complete. The operation consists in removal of parts of several ribs over the site of the gangrenous area, stitching of the pleura if adhesions are not present, and incision of the lung along the line of an exploratory trocar used as a guide, with subsequent drainage. Special precautions in regard to anaesthesia and the mode of drainage are necessary. Morriston Davies advises giving large quantities of sugar before operation, if possible.

If spontaneous evacuation occurs, resulting in cavity-formation with good drainage into the bronchi, medical measures may suffice, but if the temperature keeps up and the condition tends to become chronic, a plastic operation must be considered, as in abscess, to enable the cavity to dry up and close.

Medical measures consist in: (1) Sustaining the patient's strength by concentrated foods at frequent intervals. (2) The administration of stimulants and antiseptic expectorants. Alcohol may be given, as brandy, whisky or port wine. Ammonium carbonate, guaiacol carbonate, creosote, the balsams, and allyl compounds may be tried. (3) Attempts to evacuate the contents of the gangrenous cavity when it forms, by alterations of position and by expectorants. (4) The use of antiseptic remedies, either by inhalation or by intratracheal injection. Creosote or guaiacol, with menthol and olive oil, may be given by intratracheal injection, if the patient is not too ill. Inhalations of carbolic acid, creosote, terebene, and eucalyptol on a Yeo respirator may be tried in the endeavour to lessen the fetor. In chronic cavity resulting from gangrene, the creosote vapour bath may be employed with benefit.

Autogenous vaccines may be used as in abscess, and sometimes prove helpful.

R. A. YOUNG.

LUNG, HÆMORRHAGE OF (see HÆMORTYSIS).

LUNG, HYDATID OF (see HYDATID DISEASE).

LUNG, INFLAMMATION OF (see PNEUMONIA).

LUNG, MALIGNANT NEW GROWTHS OF

LUNG, MALIGNANT NEW GROWTHS

OF.—Tumours of the lung may be either innocent or malignant, and may originate from the connective tissue or epithelial elements. Simple tumours, of which lipoma, fibroma, chondroma, and adenoma of the bronchial mucous glands have been described, have generally only a pathological interest, except that occasionally they develop in such a way as to cause mediastinal pressure, or to impede the entrance of air to a part of the lung. We may therefore confine our attention to malignant tumours.

Etiology.—The causation is obscure. The carcinomata are commoner after the age of 40, the sarcomata in early life. There is apparently a marked preponderance in the male sex. Traumatism has been supposed to be directly connected in some cases.

Pathology.—Malignant tumours may be either carcinoma, sarcoma, or endothelioma.

Primary malignant growths in the lung are rare, *carcinoma* being more common than sarcoma. It may originate from the epithelium of the bronchi or of the mucous glands in their walls, or from the alveolar epithelium, and may therefore be either columnar or spheroidal-celled, but the cancer cells in lung growths are often strikingly pleomorphic. *Sarcoma* may originate from the connective tissue, and may be of round- or spindle-celled type. *Endothelioma* usually originates in the pleura or in the lymphatic or vascular endothelium.

Secondary or metastatic deposits of carcinoma occur most frequently from primary growths in the breast, stomach, liver, pancreas, and prostate. Secondary sarcoma is commonest from primary growth in bone.

Chorion-epithelioma and hypernephroma also frequently give rise to secondary deposits in the lungs. The lungs may be invaded by direct extension from primary growths in the mediastinum, the œsophagus, and the breast.

In primary tumours the growth is usually confined to one lung, whereas secondary deposits are frequently multiple and often distributed through both lungs. Sometimes the growth tends to spread by infiltration along the bronchi or vessels, and forms irregular masses. Occasionally large numbers of small nodules occur, constituting miliary carcinomatosis. In the course of the spread, either of primary or of secondary growths, the mediastinum or the pleura may become invaded, or pressure upon a large bronchus may be produced.

Symptomatology.—The symptoms may be

slight until the growth becomes of sufficient size to invade or press upon a large bronchus, the mediastinal structures, or the pleura. Pain, dyspnoea, loss of weight, cough and expectoration may be present, but are not characteristic. The expectoration may, however, be of currant-jelly or prune-juice character. Microscopically, the presence of groups of large cells, often containing fat, and of irregular epithelial cells is said to be pathognomonic. Some degree of fever may occur and be misleading. When there is pressure upon the mediastinum the characteristic symptoms of that condition develop. When the pleura becomes involved there is severe pain, followed by effusion: the fluid accumulates very rapidly and is usually hæmorrhagic.

Physical signs.—These are often absent until the growth is large enough to give rise to dullness, or to signs of mediastinal or bronchial pressure. On inspection, there may be bulging, if the growth is near the surface, or retraction if it has caused pressure on a bronchus. Vocal fremitus is not altered unless the growth is large, or has produced pleural effusion. Dullness is very pronounced when the growth is of large size or superficial. The breath-sounds, when present, are weak, bronchial, or stridulous, according to the situation of the growth. Adventitious sounds are equally variable, and are not characteristic. X-ray examination, especially the stereoscopic radiogram, may afford the best indications of growth.

Complications.—Bronchitis, bronchiectasis, gangrene, and pleural effusion are the most frequent.

Diagnosis.—Diseases which may give rise to similar clinical manifestations are pulmonary tuberculosis, aneurysm, pericardial or pleural effusion, and Hodgkin's disease.

In all obscure chest cases the sputum should be repeatedly examined bacteriologically and cytologically, and an X-ray examination carried out when practicable. The occurrence of pulmonary symptoms in a patient with malignant growth elsewhere should suggest the possibility of secondary deposit in the lung.

Prognosis in malignant disease of the lung is necessarily almost hopeless. The duration varies from a few weeks to two or three years.

Treatment.—Attempts have been made to remove the portion of the lung containing the tumour when the disease is recognized early, but at present surgical treatment is rarely practicable. Medically, all that is possible is to palliate the symptoms. Useless cough may

be relieved by sedative inhalations, or by a heroin or other sedative linctus. Dyspnoea may be treated by inhalation of oxygen, or by paracentesis if there is much effusion. The fluid usually re-accumulates very rapidly. Pain may be lessened by local applications, or by analgesic drugs, such as aspirin, pyramidon, phenacetin, exalgin, opium or morphine.

R. A. YOUNG.

LUNG, ŒDEMA OF.—An exudation of serous fluid into the alveolar walls and spaces. It may be chronic, acute, or hyperacute.

Etiology.—The causes are: (1) *Inflammatory*. A zone of œdema may occur around any inflammatory lesion of the lung, such as pneumonia, broncho-pneumonia, abscess, gangrene, or tuberculosis. (2) *Congestive or passive*. Any condition leading to local or general venous congestion may proceed to œdema; such are failing heart from valvular lesions or myocardial degeneration, chronic pulmonary diseases such as emphysema, and pressure from malignant disease or aneurysm. The terminal œdema of many chronic diseases and of cerebral compression is of this nature. Acute œdema may occur after the removal of a pleural effusion. (3) *Renal*. In acute and chronic nephritis pulmonary œdema may develop.

Pathology.—The main factor in the exudation of the fluid is mechanical, and depends upon the engorgement of the pulmonary capillaries. Consequently the œdema is generally most pronounced at the bases of the lungs. In renal œdema it is possible that a toxic factor is concerned and that the state of the vascular endothelium plays a part. (Edematous lung is generally dark in colour from venous congestion, heavier than normal, and on section exudes a frothy serous fluid, which is sometimes pink from bloodstaining.)

Symptomatology.—In chronic œdema the symptoms are merged in those of the primary disease, but dyspnoea and cyanosis of varying degree, together with persistent cough and the expectoration of frothy serous fluid in varying quantities, are the most characteristic features. In acute and hyperacute œdema similar symptoms develop with great rapidity. In the latter condition there is much distress, and frothy, bloodstained fluid wells up to the patient's mouth in such quantity that death from suffocation may occur in a few minutes.

The physical signs vary with the degree and extent. There are usually diminution of vocal fremitus, impairment of percussion note or

dullness, and weak breath-sounds. The adventitious sounds vary from crepitation to fine or medium bubbling or crackling râles. Rhonchi are frequently heard when the fluid reaches the bronchi. In extreme degrees of œdema both breath-sounds and adventitious sounds are absent.

Diagnosis.—It may be necessary to distinguish œdema from pneumonia, bronchitis, or pleural effusion. The symptoms and signs just described are characteristic in the absence of fever. The character of the expectoration is suggestive.

Prognosis depends upon the cause, but in hyperacute œdema it is grave.

Treatment.—This should be directed to the cause, if possible. Cardiac and renal disorders must be dealt with on ordinary lines. Engorgement of the right ventricle should be relieved by venesection, leeching, cupping, purging, or diuresis. Cardiac tonics such as digitalis and its substitutes may then be given. Oxygen may relieve the cyanosis and distress, and should be freely administered as a temporary help till other measures are effective. In hyperacute œdema immediate venesection, followed by hypodermic injection of strychnine $\frac{1}{10}$ gr. with atropine $\frac{1}{100}$ to $\frac{1}{50}$ gr., and perhaps morphine $\frac{1}{4}$ to $\frac{1}{2}$ gr., affords the most help. Later, hypodermic injections of camphor (in oil) and digitalin should be given.

R. A. YOUNG.

LUNG, SYPHILIS OF.—A specific disease of the lungs produced by the *Spiræna pallidum* either in the congenital or the acquired form of the infection.

Etiology.—The congenital form is most frequently noticed at birth or in the first few weeks of extra-uterine life. An infant, stillborn as a result of syphilis, may have many evidences of the disease, among them the white hepatization of Virchow and interstitial pneumonia. A rarer form of multiple foci due to gummata may be present either separately or, more often, coexisting with the pneumonic forms.

The acquired form occurring in adult life has been recorded as a bronchial catarrh in the secondary stage of syphilis, and as gummata as early as the second year after infection, though more commonly at later periods.

Pathology.—In the white pneumonia of infants the lung is swollen, cuts firmly, is greyish in colour owing to the exclusion, by pressure, of blood from the capillaries, and sections of it sink in water. On microscopic examination

LUNG, SYPHILIS OF

the alveolar spaces are distended with breaking-down round cells and epithelial cells.

A gumma of the lung varies in size from a microscopic lesion to one with a maximum diameter of about 2 in. It tends to produce more surrounding fibrosis than does a gumma elsewhere; it may break down and form a cavity, or it may become calcareous from the deposition of lime salts. In other respects it resembles a gumma of other organs.

Symptoms.—White pneumonia is only found in stillborn children or those who survive birth for a very short period; hence a definite clinical picture is not presented. When gummata or an interstitial pneumonia develop after birth as a result of inherited syphilis the symptoms are similar to those in the acquired form.

In the *acquired* form the condition is more elusive. Cough, expectoration, hæmoptysis, dyspnoea, pain, sweating, wasting, and even pyrexia may be present, so that the cases very closely resemble chronic pulmonary tuberculosis. It must be stated at once that, except for the absence of tubercle bacilli from the sputum on repeated examinations, the existence of syphilis of the lungs would rarely be suspected. Moreover, the absence of other clinical evidences of active syphilis in recorded cases, proved by post-mortem examinations, is noteworthy and increases the difficulties of diagnosis. The signs are more often near the hilum or even at the base, but this distribution is not rare in pulmonary tuberculosis, so the physical signs may be the same, and include congestion, consolidation, cavitation, though more often of the bronchiectatic kind, and fibrosis.

Diagnosis.—Chiefly because of the few cases seen in the post-mortem room, almost all authorities are unanimous as to the rarity of the disease. Though this is evidence that the disease is seldom of itself fatal, it hardly proves that the incidence is as rare as has been supposed. It must be borne in mind that there is a group of cases of unknown etiology closely resembling *pulmonary tuberculosis* except that the bacillus can never be found. The more extensive employment of the Wassermann reaction in these cases would betray the causation of some of them. It does not seem necessary at the present day to demand post-mortem confirmation before accepting a diagnosis of syphilis of the lungs. The diagnosis would seldom be at fault if the following four postulates were applied in cases where the

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signs and symptoms otherwise suggest chronic pulmonary tuberculosis:—

1. Tubercle bacilli are repeatedly absent from the sputum.

2. The Wassermann reaction is positive.

3. Distinct improvement in symptoms and diminution in physical signs follow treatment with salvarsan or mercury and potassium iodide.

4. The disease is permanently arrested after antisyphilitic treatment is completed.

Prognosis.—In the congenital form the disease is most serious, and life is not likely to be maintained for more than a few weeks. In the acquired form the disease can be arrested and cured, leaving a fibrosis which depends in amount on the stage at which vigorous treatment was initiated.

Treatment.—Mercury and potassium iodide must be given for the full course after intravenous treatment with a salvarsan preparation. The coexistence of pulmonary tuberculosis should not interfere with the prosecution of this treatment; the treatment of both diseases should be carried out concurrently.

A. HOPE GOSSE.

LUNG, THROMBOSIS OF (see LUNG, EMBOLISM OF).

LUNG, TUBERCULOSIS OF (see PULMONARY TUBERCULOSIS).

LUPUS ERYTHEMATOSUS.—An inflammatory disorder of the dermis, generally very chronic but occasionally running an acute course; usually confined to the face and scalp; and characterized by a tendency to produce an excessive overgrowth of the horny layer, especially in follicular openings, and to leave extensive scarring as it subsides.

Etiology.—This disease is usually one of middle life, but may begin before the age of 20, and becomes fairly frequent about the thirties. It is much more common in women than in men. The tendency in the past has been to consider it of tuberculous origin, as tuberculous lesions have been found in a high percentage of cases; lately, however, considerable attention has been directed to the streptococcus as the possible origin of the mischief, and good results have been claimed by removal of septic foci and by vaccines. It is possible that the toxins of many organisms may be capable of producing the lesions.

Pathology.—The chief change in the skin is an infiltration of the dermis in the neigh-

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bourhood of the vessels with round cells, which may destroy the hair-follicles and sweat-ducts. In the epidermis the chief change is a hyperkeratosis which is especially pronounced at the follicular openings.

Symptomatology.—Lupus erythematosus occurs in two chief types—(1) the erythematous, (2) the scaly. There is no essential difference between the two, but the first form may develop into the second, or both types may be present simultaneously; and, further, both types may be either localized or disseminated.

(1) The *erythematous type* is the less commonly met with. In its localized form it is chiefly found on the cheeks. The lesions are sharply defined, slightly raised discoid patches, circular or oval in shape, with marked infiltration, and of a red, pink, or pale-purplish colour. In the larger patches the centre is depressed and paler than the margin. The follicles are generally abnormally patulous. The onset is often acute, and then patches are apt to disappear more rapidly than the scaly ones, but leave behind well-marked atrophy. They may, however, become typical scaly patches.

The acute disseminated cases are generally of this type. In these the onset may be associated with severe constitutional manifestations, such as high fever, profound toxæmia, pneumonia, and albuminuria. The lesions are seen chiefly on the face, hands, neck and upper chest and back, and upper limbs, and sometimes develop bullæ which may contain hæmorrhagic fluid. Some cases, however, run a sub-acute course and recover, but with a liability to recurrence.

(2) The *scaly type* is generally localized, and is the common form. In this the patches are sharply defined, but often of irregular outline, and are usually slightly depressed below the surface; the colour is dull purplish-red, and the surface is covered by small yellowish or greyish scales of varying thickness. These scales are very adherent, and can only be removed with difficulty. In addition to the surface scaling, horny plugs are seen filling and dilating the follicular openings. The lesions are most commonly found on the nose, the cheeks, the helix and concha of the ears, the mastoid region, and the scalp. When the scalp is affected the hair falls out permanently. Sometimes the lesions form a continuous, butterfly-like patch on the nose and cheeks. This type also may become diffuse, and red scaly patches may develop on the hands,

especially the back of the fingers, also on the forearms, arms, and upper part of the trunk (PLATE 17). Patches are sometimes seen on the mucous membranes, chiefly on the vermilion border of the lips, on the inner aspect of the lips, and on the palate.

Diagnosis.—The acute erythematous form must be distinguished from erysipelas and erythema multiforme. In lupus erythematosus the lesions are usually symmetrical, persistent, and may scale, while in *erysipelas* they are asymmetrical and tend to clear up in one place and spread in another. In *erythema multiforme* the lesions are less persistent than in lupus erythematosus, and tend to affect the extremities more than the face and to disappear without scarring. The chronic scaly form has to be distinguished from *lupus vulgaris*. In this condition the typical lupus nodules are present, and there is a tendency to ulceration which is absent in lupus erythematosus; further, the latter occurs more or less symmetrically on the face and usually begins later in life than the former.

Prognosis.—The prognosis of the *acute* form is generally bad, though some patients recover. A considerable number, however, die in a septicæmic state or from pneumonia or some other intercurrent condition.

The *chronic* forms are very resistant to treatment, and recurrences are frequent even when the patches subside. The disease may last for years, and, even when it clears up, often leaves disfiguring scars and patchy loss of hair.

Treatment.—In the *acute* cases everything depends on the general treatment. The patient should be put to bed, and any general condition treated. When septicæmia is present, cultures from the blood, faeces, and urine should be made and antistreptococcal serum or vaccine administered as necessary. Quinine and the salicylates in large doses have also proved of considerable value in these cases.

In the *chronic* cases an attempt should first be made to discover any focus of disease, such as tuberculosis, local forms of sepsis, pyorrhœa, or tonsillar infection, and appropriate treatment adopted. Quinine has proved beneficial in chronic as well as in acute cases, and in some cases arsenic is valuable.

Local treatment should be sedative in the erythematous and more acute scaly varieties, calamine lotion, applied frequently, being the most useful. In cases of longer standing more vigorous treatment is required. The patches can be washed with soft soap and an ointment



PLATE 17.—LUPUS ERYTHEMATOSUS OF FACE, TRUNK,
AND ARMS.

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of salicylic acid 2-4 per cent., or pyrogallol acid 10 per cent., applied for a day or two, and the reaction produced allowed to subside under calamine lotion. Applications of CO₂ snow, for 5-15 sec., often have a beneficial effect. Painting with pure carbolic acid or, as lately recommended, rubbing in carbolic acid 1 part, strong lactic acid 4 parts, after cleansing the patches with ether, has produced good results in some cases. X-ray-therapy has proved disappointing, but good results have been claimed for ultra-violet light.

A. M. H. GRAY.

LUPUS VERRUCOSUS (see under Lupus Vulgaris, in SKIN, TUBERCULOSIS OF).

LUPUS VULGARIS (see SKIN, TUBERCULOSIS OF).

LUTEIN CYSTS (see OVARIAN CYSTS).

LYMPH SCROTUM (see FILARIASIS).

LYMPHADENITIS (see LYMPHATIC GLANDS, ENLARGEMENT OF; LYMPHATIC GLANDS, TUBERCULOSIS OF).

LYMPHADENOMA (syn. Hodgkin's Disease, Lymphomato-s Granulomatosa, Lympho-granulomatosis Maligna, Adénie).—An infective adenitis, at first local and subsequently generalised, with secondary anæmia and a fatal termination.

Etiology.—The condition is frequent in early adult life, and attacks males more often than females. A primary focus in the tonsils, mouth, or elsewhere often exists. Several authors have described a minute bacillus, such as an acid-fast organism regarded as a modification of the tubercle bacillus (Fraenkel and Much); and recently a strong claim has been made for a pleomorphic diptheroid bacillus (*Bacterium hodykini*) which is Gram-positive and not acid-fast (Bunting and Yates). Sternberg's view—now abandoned—that the disease is a modified form of tuberculous lymphadenitis was due to examination of lymphadenomatous glands infected with tuberculosis. Implantation of lymphadenomatous glands into animals has been negative, but inoculation of virulent cultures of *Bacterium hodykini* into monkeys has produced lesions of the lymphatic glands. D. Symmers believes that lymphadenoma is neither an infective granuloma nor a neoplasm, but a systemic disease with a predilection for lymphoid tissues, and that, as a result of proliferative changes in the

bone-marrow, cells are transported to the lymphatic glands and are responsible for the changes there.

Morbid anatomy.—The enlarged lymphatic glands are either soft or hard according to the amount of fibrosis. At first separate, they become united by peradenitis into packets, and in chronic cases there may be much peradenitic fibrosis. On section, the glands are greyish-white in colour, and are not caseous unless tuberculous infection has supervened. Those in the posterior cervical triangle are first and most often affected, then extension occurs to the axillary glands on the same side, the opposite cervical region, the mediastinum, the opposite axilla, the groins, the spleen, and the abdominal glands. There may thus be continuous chains of enlarged glands from the neck into the chest, or along the spine and aorta. Individual glands may be as large as a hen's egg, and the packets of adherent glands may reach the size of a coconut.

Microscopically, the whole gland is altered and homogeneous; the lymphocytes are diminished and no longer conceal the framework of the gland, which undergoes hyperplasia. There is an increase of the endothelial cells and of the cells of the reticulum, some of which attain a large size and contain four or more centrally-placed nuclei; these are the "lymphadenoma cells," which differ from the giant cells of tuberculosis. Eosinophil cells are always present, sometimes but not always in greatly increased numbers. This local eosinophilia is most marked in acute cases, and has been explained as due either (a) to immigration from the bone-marrow, or (b) to a local production on the ground that their nuclei differ from those of the hæmic eosinophil cells (Turnbull). Lymphadenoma does not show the same structure at all stages. In the early stages there is hyperplasia of lymphoid cells and the lymphadenoma cells are absent or rare, and the later stages show a great preponderance of fibrous tissue. At these two extremes there may hardly be sufficient histological evidence to diagnose the condition as lymphadenoma. The difference between the soft and hard forms is merely one of duration. When tuberculosis supervenes the two lesions can be distinguished from each other.

The spleen, which is more or less enlarged in 75 per cent. of the fatal cases, often shows pericapsular adhesions and sometimes slight irregularities on the surface. It preserves its shape, and is firm on section, with white

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grey masses which closely resemble unsoftened caseous tuberculomas, an appearance long ago described as the "hardbake spleen." Microscopically, the white areas show the same structure as the affected lymphatic glands, and the rest of the organ may exhibit considerable fibrosis.

The liver may be enlarged from perivascular infiltration with lymphoid tissue, which Adami regards as a compensatory hyperplasia to make up for the destruction of lymphatic tissue elsewhere, and not as a metastasis. Subsequently, however, this compensatory tissue may become lymphadenomatous.

Symptomatology.—Cases may be divided into *acute* and *chronic*, a special variety of the latter being the *relapsing* form. The acute cases are very rare, and in some so described the disease has probably existed unnoticed until generalization occurred. The acute cases, which are febrile and may begin with tonsillitis, must be distinguished from acute leukaemia.

The term *latent* has been applied to two groups of cases: (a) those without palpable glands but with constitutional symptoms and even fever of the relapsing type, and (b) those with little glandular enlargement, abeyance of symptoms and a long course.

The usual course is chronic; for a long time enlarged glands in the neck may be the only manifestation of the disease. Later, the morbid process extends in the order described above, and eventually generalizes. The clinical manifestations are (a) general or constitutional, and (b) local or due to pressure exerted by the enlarged glands.

Temperature.—The early stage of chronic lymphadenoma is free from fever, but most cases show it at some time or other, usually late in the disease. At least three forms of fever occur: (1) a persistent mild form, usually 101–102° F., with a daily variation of 1.5°—this may last for months; (2) high irregular temperature with morning remissions to 100° F.—this is seen in the late stages, and may be accompanied by rigors and sweating, thus imitating suppuration; (3) a remarkable relapsing form (Pel-Ebstein recurrent fever) with alternating febrile and afebrile periods of about 5 to 15 days each, but considerable variations occur. This form may be associated with enlargement of the external and the internal lymphatic glands, or of the internal only. The febrile periods are usually accompanied by much constitutional disturbance and by further enlargement of the lymphatic glands

and the spleen, which recedes in the intervals. An abnormal appetite has been noted in the afebrile periods. This relapsing fever has been known to continue for a year or even more, but its average duration is 7½ months (Batty Shaw). It persists until death. Some cases of tuberculous adenitis and of chronic pulmonary tuberculosis closely imitate it. Although it has, naturally, been suggested, there is no convincing evidence that the relapsing fever is due to a superadded infection: it has recently been thought to be anaphylactic, and by D. Symmers as the result of absorption from multiple focal necroses, especially in the spleen.

Blood.—It is generally stated that there is nothing diagnostic about the blood, but according to Bunting's recent observations there is a constant increase in the blood-platelets and the transitional cells. In addition, cases of under a year's duration show a relative lymphocytosis, a diminution in the eosinophils, and no leucocytosis, whereas in more advanced cases when the disease is generalizing there is usually a leucocytosis of about 20,000 with a high (76.90) percentage of polymorphonuclears. In quite exceptional cases there is considerable eosinophilia. As the disease advances, secondary anaemia appears, and later, from exhaustion of the bone-marrow, there is an absence of blood-platelets.

Skin.—Bronzing is occasionally seen in patients who have not taken arsenic or been treated by X-rays, and may be due to irritation of the sympathetic ganglia or system. Itching of the skin without a local cause (pruritus) is rare, probably occurring in about 5 per cent. of the cases. It may be an early symptom before glandular enlargement is obvious, or may occur late in the disease, and may be so intense that open sores are caused by the scratching; it may pass off permanently, or may recur and may then correspond with periodic enlargement of the superficial glands; it is probably toxic. Itching may be associated with eruptions such as prurigo, papules, or general exfoliative erythrodermia, which are probably all toxic in origin and not characteristic of lymphadenoma. Infiltration of the skin by lymphadenoma is rare; it occurs as purple or slate-grey discoid infiltrations, and is not necessarily associated with pruritus. It must be distinguished from cutaneous sarcoma, leukaemic infiltration, mycosis fungoides (in which also pruritus precedes any obvious tumour), and from Kaposi's lymphodermia perniciosa. Erythematous and bul-

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lous eruptions are very occasionally seen, and herpes has been induced by the arsenic so commonly given in the disease. Purpura is very rare.

The **lymphatic glands** may slowly increase or diminish in size; in some instances the superficial glands undergo fibrotic contraction while the internal glands are enlarging. In the relapsing type of fever the glands may rapidly enlarge during the febrile periods, and recede in the intervals; and this enlargement, which appears to be due to exacerbations in the peradenitis rather than to swelling of the glands themselves, may be accompanied by boggy and redness of the overlying skin. Enlarged glands in the neck may exert pressure on the trachea or produce oedema of the aryteno-epiglottidean folds. In the thorax, lymphadenomatous glands may behave like a mediastinal growth, and by pressure on the bronchi or recurrent laryngeal nerve set up painful dyspnoea and cyanosis. The large veins may be compressed and cause distension of the subcutaneous veins. In rare instances there is dysphagia. Serous or, less often, chylous pleural effusions may depend on venous or lymphatic obstruction; and sudden death has occurred in such cases or as the result of pericardial effusion. In very exceptional instances there is extensive intrathoracic lymphadenoma without other glandular enlargement.

Enlarged intra-abdominal glands may give rise to pressure on the portal vein and ascites, on the bile-ducts and jaundice, on the inferior vena cava and oedema of the feet, on the ureters, on the lumbar and other nerves, causing pain. Ascites may be serous, chylous, or pseudochylous. Enlargement of the retro-peritoneal glands, when accompanied by fever, provides a very difficult diagnostic problem; when in addition the spleen is enlarged, malaria and enteric fever must be eliminated by a blood examination.

In exceptional instances lymphadenoma chiefly, if not entirely, attacks the spleen and simulates splenic anaemia or, if there is ascites, Banti's disease. When the liver is much enlarged and fever is present, hepatic abscess may be suspected if glandular enlargement is absent or overlooked. Such hepatomegaly can be distinguished from that of syphilis by the Wassermann reaction.

Death may be due to cachexia with grave anaemia, to obstruction exerted by enlarged glands on the trachea, bronchi, great vessels, or thoracic duct, or to effusion into the serous

cavities. Tuberculosis or acute infections may prove fatal.

Diagnosis.—To be absolutely certain, microscopic examination of an excised gland is necessary, for *tuberculous adenitis*, particularly the form with large-celled hyperplasia and little or no caseation, may simulate exactly. Although widespread glandular enlargement is, generally speaking, in favour of lymphadenoma, caseous tuberculous adenitis is sometimes similarly distributed. As tuberculosis may complicate lymphadenoma, undue weight must not be attached to a positive tuberculin reaction. Examination of the blood will exclude *lymphoid leukaemia*, and the Wassermann reaction will be of assistance in eliminating *syphilis* in cases with a large spleen and comparatively little glandular enlargement, in which this question may arise. In very rare instances splenomegaly may be the only palpable manifestation of lymphadenoma, and the condition may resemble chronic *splenic anaemia*. In the cases best described as *pseudo-leukaemia*, in which the glands are enlarged but do not show the histological changes of lymphadenoma, and in which the blood is not leukemic, microscopic examination of a gland is the only means of diagnosis. As true lymphadenoma seldom or never attacks the intestines, whereas pseudo-leukaemia does, diarrhoea is in favour of the latter. The diagnosis from *lympho-sarcoma* may be extremely difficult, especially when the disease is mainly intrathoracic or abdominal and it is impossible to remove a gland for examination. In rare cases widespread *malignant disease* may simulate lymphadenoma, especially when a left-sided hypernephroma imitates a large spleen and gives rise to growths in glands above the clavicle and elsewhere. I have seen a case of *neuro-fibromatosis* which was regarded as lymphadenoma. The cutaneous lesions have been mistaken for prurigo and scabies.

Prognosis.—True lymphadenoma is generally regarded as invariably fatal, but possibly this view may require modification if wide removal of the local focus at an early stage and immediate excision of any recurrence are carried out systematically. The duration of survival after the onset of symptoms varies considerably; the rare acute cases may terminate within a few weeks or months, whereas the chronic cases usually prove fatal within three years. Out of Longcope's 49 carefully selected cases, 34, or 69.4 per cent., died within

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two years, but 2 survived for seven years and another for six years. The application of radium or of X-rays certainly prolongs life. The prospect of life varies according to the virulence and site of the disease; slowly-growing glands which remain discrete, even though present in more than one position—for example, in both sides of the neck and one axilla—are more favourable than glands which coalesce and become adherent to the skin from repeated attacks of periadenitis, even though one supraclavicular fossa only is obviously affected, for the latter type is especially prone to invade the mediastinum and disseminate widely (Yates). The intrathoracic and abdominal forms of lymphadenoma are more rapidly fatal than widespread superficial glandular infection, and mediastinal lymphadenoma may prove fatal by mechanical pressure on the bronchi or great vessels. The cases with relapsing fever, more especially those in which the internal glands are affected, run a more rapid course than others. At a late stage, when the disease has generalized widely, the end is near, especially in the presence of grave anaemia and high fever; but even then in exceptional cases life may be prolonged much more than appears probable. The disappearance of obvious glandular enlargement is not necessarily a good sign, for the disease may be actively advancing internally. Further, even when the amount of lymphadenoma has been greatly reduced by means of X-rays, the patient may pass into a condition of fatal cachexia and extreme anaemia. The onset of acute infections or of tuberculosis renders the outlook most unfavourable.

Treatment.—Prophylaxis consists in the removal or cure of foci in the mouth, nose, or ears which might lead to glandular infection. Thus, large tonsils should be removed and oral sepsis treated.

Medical treatment.—It is generally agreed that arsenic, especially in the early stage, may reduce the size of the glands and apparently bring about an intermission, but a relapse occurs sooner or later. Arsenic does not, however, succeed in all cases, and in other cases, though successful at first, it fails in a relapse. In this connexion the view that the disease may undergo a transformation into sarcoma should be mentioned. Other arsenical compounds, such as atoxyl and orsudan, have been tried. Salvarsan may produce very considerable benefit, which unfortunately appears to be temporary only. The patient

should be placed under the best hygienic conditions, and depressing factors—which, by diminishing resistance, favour recurrence—avoided as far as possible. X-rays diminish the size of soft glands and the spleen, but do not affect fibrotic glands, and probably do no good in the form with relapsing fever. Pancoast's statistics show that 25 per cent. of the cases treated by the rays are greatly benefited and are well three years after treatment. On the other hand, relapses are common, sometimes soon after the treatment ceases, and grave toxæmia may be induced by it. Normal lymphatic tissue is destroyed by X-rays, and, if this is carried to excess, harm will result. The leucocyte count should therefore be watched during treatment. Radium exposures give better results than X-rays and should be brought to bear on all the lymphatic areas, including those in the abdomen and chest, and not confined to the palpable disease (Simmons and Benet). Improvement becomes obvious in one to three weeks after the treatment is begun. Neither X-rays nor radium can be said to cure the disease.

Vaccine treatment with *Bacterium hodgkini*, beginning with 5 millions and increasing gradually with five to seven days' intervals to 100 millions, has not justified the encouraging results first reported by Billings and Rosenow. An immune serum (to the diphtheroid organism) is on trial (Bunting).

Surgical treatment.—The complete removal of all the disease in an early stage when it is localized is a logical form of treatment, and recently it has been urged that an extensive operation on the lines recommended for carcinoma by Halsted and Crile should be performed, the exposed parts being freely treated with tincture of iodine to obviate contamination. After the operation repeated exposures to X-rays should be carried out. Any recurrence should be widely removed at the earliest possible date. Incomplete operation is sometimes followed by rapid recurrence, and, generally speaking, the results of surgical interference have been so disappointing that this form of treatment has been viewed with some disapproval. But possibly the more radical measure outlined above, if carried out at an early date, may lead to a change of opinion. Partial excision of glands should certainly not be attempted except to relieve pressure symptoms or perhaps to remove deformity.

H. D. ROLLESTON.

LYMPHATIC GLANDS, ENLARGEMENT OF

LYMPHANGIECTASIS (see Lymphatic Nævus, under NÆVUS).

LYMPHANGITIS (see CELLULITIS).

LYMPHATIC GLANDS, ENLARGEMENT OF.—The problem presented to the clinician by an enlargement of the lymphatic glands varies according to (1) the situation of the glands, (2) the history of the enlargement, (3) the age of the patient, and (4) the physical characters of the enlargement.

1. **Site.**—The groups of lymphatic glands accessible to the examiner are the cervical, submaxillary, axillary, and inguinal, and in a less degree the intrathoracic and intra-abdominal. Besides these, there are other glands more or less isolated, enlargement of which is sometimes of considerable importance in diagnosis; for example, the preauricular, the parieto-thoracic, clavicular, and cubital glands.

2. The **history** of the enlargement is of the first importance. If it has been sudden, the most probable cause is an acute infection, due either (a) to some generalized disease (hæmatogenous infection), or (b) more commonly to a local bacterial invasion of the tissues (lymphogenous infection). If, on the other hand, the enlargement is insidious in its onset, and progressive, the field of possible causes is much extended.

3. The **age** of the patient affords valuable help. In children and adolescents the likelihood of acute infection, either hæmatogenous or lymphogenous, is considerable, whilst in middle life and old age, cancerous enlargements form a fairly large proportion.

4. **Physical characters.**—Lastly, the physical characters of the swelling must be carefully investigated: the situation; whether it corresponds to the known position of glands; the mobility or fixation of the tumour, both to the skin and underlying tissues; the hardness, elasticity, fluctuation, tenderness, and coalescence of the individual glands—all must have their due appreciation.

Causes. Cervical glands.—Of the causes of acute enlargement, inflammation due to the invasion of organisms, or their toxins, is by far the commonest, and the glands of the neck are the most often affected. This evil prominence they owe to the frequency of acute infections of the mouth, fauces, tonsils, and ears, and of bacillary or parasitic affections of the scalp and face. Thus, enlargement of the deep cervical glands is usual in the sore throat of scarlet fever and diphtheria; in the

suppurative affections of the tonsillar fossæ in adolescents and adults, and of the lateral and posterior pharyngeal walls in infants. In no case where there is acute enlargement of the cervical glands in an infant should a careful examination of the pharyngeal walls be omitted. In school children pediculosis and impetigo of the scalp and face are frequent sources of infection, and the submental gland situated in the mid-line is enlarged only from that cause, or from an ulcer in the floor of the mouth. Otitis media, again, especially of the more chronic type, is often ignored by careless persons till the glandular swelling excites their fears by deforming their appearance. A similar observation is true of alveolar periostitis and its sequelæ. A specially severe infection of the cervical, submaxillary, and submental groups, spreading to the neighbouring tissues, is known as Ludwig's angina (q.v.).

Of the more chronic cervical enlargements, the most important is the result of infection with the *Bacillus tuberculosis*. (See LYMPHATIC GLANDS, TUBERCULOSIS OF.)

Lymphadenoma (q.v.) often begins in the cervical group, and must be recognized by a process of exclusion, or by direct examination of a gland excised for the purpose. Enlargements due to lymphocytic leucæmia or chloroma or sarcoma are rare.

Syphilis is a cause of enlargement not only in the hæmatogenous infection of the secondary stage, but also in the congenital disease, and late in the tertiary stage. Its recognition, apart from microscopical examination, is difficult. The most serious form of enlargement after childhood is that due to metastatic deposits of carcinoma, the primary seat of which is commonly in the mouth, tongue, pharynx, larynx, or œsophagus. Nor must the possibility of primary new growth of the glands be forgotten. Such growths are rare; in children they are usually sarcomata of the round-celled type; whilst in adults there occurs a peculiar large-celled endothelioma, of slow growth, and often of slight local malignancy, but prone to metastasis, so that successful removal of the local swelling often does not remove the disease.

Immediately above the clavicle there are small groups of glands which on the left side are apt to become enlarged by metastasis from a carcinoma of the stomach, a fact which is sometimes of considerable aid in diagnosis.

Dealing with the other groups briefly, the axillary lymphatic glands suffer chiefly from

LYMPHATIC GLANDS, TUBERCULOSIS OF

acute infections, tuberculosis, or lymphadenoma, or from carcinomatous deposits from a breast cancer. The **inguinal group** are chiefly liable to acute infections, especially of the penis or vulva, or of the feet and legs; they are also sometimes lymphadenomatous, but in many cases of this disease are not involved. Mention must also be made of the axillary and inguinal "buboes" of plague. The **pariethoracic glands** are said to drain the pleural membranes, and their enlargement may thus offer an indication of tuberculous or pyogenic infection of the pleural cavity. The **cubital glands** often escape bacterial invasion when the axillary are affected, and, on the whole, are rarely enlarged. Tuberculosis, syphilis, lymphadenoma, rubella, and glandular fever are all possible causes.

Of enlargement of **intrathoracic glands**, all that need be said here is that, apart from tuberculosis and new growths, the enlargement is rarely sufficient to give rise to physical signs. Very occasionally pyogenic cocci cause suppuration in these glands, and produce a mediastinal abscess. Of enlargements of **intra-abdominal glands**, those which give rise to clinical symptoms are most often tuberculous in origin (see LYMPHATIC GLANDS, TUBERCULOSIS OF), but occasionally masses of glands enlarged by secondary new growth form palpable tumours.

Summing up the difficulties, it may be said that in the absence of evidence of acute disease, either hæmatogenous or lymphogenous, the differential diagnosis in the child must be made between tubercle, syphilis, and lymphadenoma; whilst in the adult there is the added difficulty of carcinomatous enlargement. In the absence of definite indications, excision and microscopical examination will usually determine the point.

HUGH THURSFIELD.

LYMPHATIC GLANDS, TUBERCULOSIS OF.

Of all the causes of enlargement of the lymphatic glands, tuberculosis, by reason of its great frequency, is the most important. In the majority of instances it is a local (lymphogenous) infection, but in rare instances a widespread enlargement of many glands occurs, and is possibly due to a hæmatogenous infection.

Frequency.—The frequency of tuberculosis of the glands can hardly be exaggerated, in view of the knowledge we have derived from the careful examination of morbid anatomical specimens; it is at least probable

that every adult in the dense populations of civilized countries has had and has overcome the infection. On the other hand, it is easy to exaggerate the frequency with which this infection gives rise to clinical symptoms, or to visible or palpable tumours; and it is quite certain that enlarged glands commonly said to be tuberculous, especially in the cervical region, are often found to be non-tuberculous when examined microscopically.

Etiology.—Congenital tuberculous infection is so rare as to be clinically negligible, and hence we find that in the majority of cases of tuberculosis of the glands there is a source of infection in close relation to the victim. The two chief sources undoubtedly are (1) contact with infected persons, (2) ingestion of infected milk.

Human and bovine infection.—The relative proportion of human and of bovine tuberculous infections is still a matter of contention, but it would appear from the most recent careful researches that, taking all forms of tuberculosis together, the form which exhibits the largest percentage of bovine infection is glandular tuberculosis, but that even here the proportion is not more than one-third, and that the infected human being remains by far the most serious focus of dissemination of the disease.

The age at which tuberculosis of glands is chiefly met with is in childhood from 2 to 15 years, though no age is exempt, and the age-incidence varies somewhat with different groups of glands. For example, tuberculosis of the axillary glands occurs usually in adults or adolescents, rarely in children.

Clinical tuberculosis of the lymphatic glands is most frequent in the cervical, bronchial, and mesenteric groups; while the axillary and inguinal groups are much more rarely involved.

Clinical symptoms and course. 1. **Generalized glandular tuberculosis.**—This rare affection is met with at all ages, but most commonly in children. The onset of the illness is usually abrupt, with a moderate degree of fever and rapid wasting. The most prominent feature of the disease, however, is the rapid simultaneous enlargement of all the superficial lymphatic glands, which are swollen, tender, and sometimes "softening," causing redness of the overlying skin. Occasionally the glandular infection spreads to other tissues, and the illness runs the course of an acute miliary tuberculosis, but more commonly, after a period of acute inflammation the swellings subside, the

glands harden, some of them become caseous, and the patient recovers health and strength. He is, however, liable to reinfection from these glands, or to the formation of sinuses.

2. Local glandular tuberculosis.—The more usual type of glandular tuberculosis is by local infection—the enlargement of a single group of glands, or even of a single gland. At first the swelling may be tender and painful, more often it is almost painless, and can be handled freely. The glands form separate rounded firm elastic swellings, of various sizes; at first freely movable beneath the skin and on the deeper structures, but later prone to become fixed, and to involve the overlying skin. It is uncommon to find but a single gland enlarged; more usually all of one group are attacked, but in various degrees, so that at a given time the practitioner may find glands of all sizes, all degrees of firmness, mobility, and elasticity. The progress of the affection is variable, according to the degree of resistance of the patient's tissues. In the most favourable case the tuberculous focus becomes surrounded by fibrous tissue, degenerates, and eventually calcifies. Such a gland is probably no longer a source of danger to its possessor, although it is rare for all the glands of a group to have reached this stage. More commonly the lesions are found in different stages of development, and while the infection is stationary or retrogressive in one gland it is extending in another. When a gland becomes wholly caseous it may "soften." Frequently this softening is the result of secondary infection with pyogenic organisms, but it appears that softening can occur without this intervention. The softening leads to the inflammation of the tissues surrounding the gland, and often eventually to the formation of a sinus through the overlying skin. The glands of the cervical group are perhaps more prone to undergo softening than those of other groups, probably because the area which they drain is often the seat of pyogenic infections, but bronchial and mesenteric glands may follow the same course.

Usually, when the glands only are involved, there is but little disturbance of the patient's general health, at least until the softening process begins. The exceptions to this rule are chiefly found in the intrathoracic and intra-abdominal groups, where pressure upon important structures may give rise to serious symptoms.

Diagnosis.—In the generalized type of the

disease there is, as a rule, no difficulty, since there are but few complaints in which many groups of lymphatic glands are simultaneously enlarged. Lymphadenoma has been a cause of error, and in one case the enlargement due to syphilis was confidently diagnosed as tuberculous. Microscopic examination of an accessible gland will always settle the diagnosis.

In the local type of the disease the diagnosis of an enlargement must rest upon a full consideration of many factors; the age of the patient, the history of the swelling, the general symptoms, and lastly the physical characters of the swollen glands. Thus, in a child with swollen cervical glands, who has an obvious source of infection with pyogenic organisms, such as chronically inflamed tonsils, in whom the general health is more or less seriously disturbed, and often with periods of fever, the probability is strongly against the glands being tuberculous; whereas when the glands are enlarged insidiously, with little or no disturbance of general health, and an absence of obvious foci of sepsis, a diagnosis of tuberculous infection is far more probable. In each individual case the diagnosis must be conducted upon similar lines, for with rare exceptions the various pathological aids, such as *v. Pirquet's* or *Calmette's* test, or the subcutaneous tuberculin reaction, afford no assistance in this sphere. Mistakes are, however, not common, if it be remembered in the case of cervical enlargements that no diagnosis of tuberculosis is justifiable before all septic foci in the drainage area of the enlarged glands have been cleared out.

Besides the enlargement due to chronic pyogenic infections, there are two other diseases which may lead to confusion—syphilis and lymphadenoma. There occur from time to time examples of cervical enlargements more or less insidious in onset, resembling in their physical characters tuberculous enlargements but nevertheless of syphilitic origin. Such instances are not common, and can be diagnosed only by attention to the history and to the general characteristics of the patient's illness, by the Wassermann reaction, and lastly by the incision of an accessible gland. The same is true of the more chronic lymphadenomatous swellings, though in this case the physical characters of the glands themselves often give some aid; the enlarged glands tend to be more uniform in size, less intimately attached to one another, and more elastic to the touch. In older patients,

and very occasionally in young children, the question of malignant disease—carcinoma or sarcoma—must be considered, but the opportunity for error here is not great, if the history of the swelling be carefully considered.

The same considerations hold good in the case of *axillary* or *inguinal* swellings, it being remembered that tuberculosis in these sites is more often met with in adults or adolescents than in children before puberty.

The diagnosis of *intrathoracic glandular tuberculosis* is most uncertain. There can be no doubt that where there is collapse of a part of the lung, usually the left upper lobe, combined with an area of impaired resonance in the interscapular region, there is, in the absence of other indications, a presumption that enlarged glands are pressing upon the bronchus. Such a combination of symptoms and physical signs is found occasionally in children, very rarely in adults, as the result of tuberculous enlargement of the bronchial glands; but, speaking generally, such signs are rarely present except in children in whom the disease has already made serious inroads on the lungs as well as on the glands that is, when the disease has already advanced beyond the stage of glandular tuberculosis. An X-ray examination may confirm the suspicions excited by the physical signs, but the bronchial glands are so situated, and the degree of enlargement sufficient to cause pressure is often so slight, that the skiagram seldom gives much help.

Intra-abdominal gland tuberculosis presents itself chiefly in the form of abdominal tumours. The tumour is situated usually in the right or left iliac fossa, and can be recognized by its uneven surface, its immobility on the deeper structures, and often by its palpability per rectum. Isolated mesenteric glands also sometimes cause trouble chiefly in the form of attacks of abdominal pain, probably due to spasm, to contracture, or to kinking of a part of the intestine. Appendicitis, intussusception, and faecal accumulations are the chief sources of error, and can usually be distinguished by attention to the history, and to the characters of the tumour.

Treatment.—The treatment of glandular tuberculosis will vary with the individual case, according to the situation, stage of development, and general characters of the swelling. In all cases the general treatment must follow the lines of the treatment of tuberculosis of any organ; that is, a maximum of air and

sunshine, nourishing food, and attention to the smallest details that make for health. In addition, in the case of cervical-gland tuberculosis, it cannot be too strongly emphasized that all sources of pyogenic infection, such as carious teeth, chronically enlarged tonsils, or adenoids, must be carefully removed. In spite, however, of the greatest care, a number of patients will require removal of the glands by operation, and though it is often not possible by this means to remove all the infected tissues, yet the prospects of recurrence of a tuberculous tumour in the same region are slight. The injection of tuberculin is advocated by some authorities, decried by others. On the whole, the sum of authority would appear to indicate its employment on the ground that it is at least harmless, and in some instances appears to expedite resolution. It is also advisable to use it after removal of tuberculous glands, since it obviates the tendency to thickening of the scar. The doses should be those recommended for the treatment of other forms of tuberculosis.

Some of the most difficult problems of therapeutics are presented by tuberculosis of the intra-abdominal glands. Operative interference with these tumours has often been attended by most brilliant success, but, on the other hand, it is not uncommon to find, on opening the abdomen, that successful interference is impossible, and in a few instances even successful operation has not evaded the risk of infecting the abdominal wound and producing a sinus. Each case must be judged on its merits, but, unless the abdominal tumour is causing trouble from pressure, it is advisable to give an extended trial to other therapeutic measures before resorting to operation.

Removal of the axillary and inguinal groups is generally easier, and more likely to be complete; moreover, the original focus of the disease is more readily detected and dealt with.

HUGH THURSFIELD.

LYMPHATISM.—Lymphatism, or status lymphaticus, is said to be present when the lymph-glands and lymphatic structures of the tonsils, pharynx, and alimentary tract are obviously enlarged, and the spleen and thymus are hypertrophied. It is thus obviously a pathological term, and its transference to clinical medicine has given it meaning an obscurity and vagueness which defy definition. Paltauf employed it to denote the condition which he observed in the bodies of infants who

LYMPHATISM

had died suddenly and unexpectedly; and since the most striking feature in these cases is the enlargement of the thymus, in medicine the term has become applied to all cases where this organ is definitely enlarged.

Etiology.—The chief sufferers from lymphatism are infants, but, in the broad and loose sense, no age is exempt. The enlargement of the thymus, the hypertrophy of the lymphatic structures of the fauces and of the tongue, the prominence of the solitary follicles of the intestine, and of the Malpighian follicles of the spleen are found (1) in infants dying suddenly (thymus death); (2) in children and adolescents dying unexpectedly under anæsthetics, especially chloroform; (3) in certain types of mental or physical defectives; (4) in infants who have suffered from continuous or spasmodic dyspnoea (thymus asthma).

Symptoms.—The condition generally remains undetected during life; but anæmic children who have flabby muscles and are easily tired, who are subject to attacks of dyspnoea, or have obvious lymphatic hypertrophy, such as large tonsils, adenoids, large cervical glands, or an enlarged spleen without obvious cause, may be suspected. An examination may then lead to the detection of enlargement of the thymus gland, either as a distinct tumour in the suprasternal fossa, or by an increased area of dullness to percussion over the manubrium sterni, or by X-ray demonstration. Thymic dyspnoea is said to be of a characteristic nature, but is certainly a rare phenomenon in this country. More usually the disease is brought to the notice of the practitioner by the sudden death of an infant who has apparently been in robust health, or by the death of a child who for some trifling operation has inhaled chloroform. The cause of the condition is unknown, nor is it known what is the connexion between the sudden failure of vitality and the lymphatic hypertrophy.

Diagnosis.—When the desirability of giving an anæsthetic to a child is in question, the possibility of the lymphatic state should always be borne carefully in mind; but diagnosis in the ordinary sense is, so far as can be said at present, impossible, the only direct evidence being the dull area of an enlarged thymus gland together with a skiagram showing the enlargement. When such signs are present an anæsthetic should be avoided, if possible, and apparently chloroform should never be used.

LYMPHO-SARCOMA

Treatment.—The manner of the death suggests that it is due to a sudden fall in blood-pressure, and hence it is advisable that the anæsthetist should have at hand such drugs as pituitrin, $\frac{1}{2}$ c.c. of which should be used in case of necessity.

In cases where the anæsthetic has been postponed because the disease is suspected, the main lines of the treatment should be abundance of open air and exercise, with such drugs as iron and arsenic, and careful attention to the digestion. Massage of the flabby muscles is also probably of considerable service.

For the dyspnoea attributed to the presence of an enlarged thymus gland, operative interference has been advocated and carried out both in America and Germany. The results, however, can hardly be regarded with enthusiasm, nor is it as yet by any means clear that the dyspnoea is due to the presence of the enlarged gland. In the present state of our knowledge it is certainly preferable not to resort to thymectomy except in cases of extreme urgency.

HUGH THURSFIELD.

LYMPHOCYTHÆMIA (see LEUKÆMIA).

LYMPHOCYTOSIS (see LEUCOCYTOSIS).

LYMPHO-SARCOMA.—A tumour whose characteristic element is a round cell, resembling the round cell of the lymphatic gland, and whose structure closely simulates that of the lymphatic gland. Unfortunately, the term is applied to almost any round-celled sarcoma, but it should be strictly reserved for those growths which, so far as can be ascertained, have their origin in lymphatic tissue.

Pathology.—Tumours of this type are commonly malignant in the highest degree; not only do they spread rapidly at their original site, pushing aside or destroying the adjoining tissues, but they disseminate widely, and the metastatic growths appear thickly in the viscera or bones. They occur at any age, but are distinctly more common in childhood and adolescence than in adult life.

Clinical course.—Their principal situations are the neck and the mediastinum. In the neck, the tumour makes its appearance usually in the deep cervical glands of one side, involving the whole group with great rapidity. The glands are at first discrete, but quickly become matted together, firm, and elastic. They are very little, if at all, movable on the deeper tissues, but do not as a rule involve the skin. By the time that the tumour

MADURA FOOT

has attained a size which begins to press upon adjacent structures, the metastatic growths in the pleural membranes, in the lungs, liver, or long bones are so far advanced that the patient is in a cachectic condition, and survives but a very short time. Even shorter is the prospect of life when the mediastinal glands are the primary seat of the disease. The pressure upon important structures in the thorax, and the early involvement of the lungs, produces a condition of dyspnoea from which no relief can be obtained. The patients often die of cachexia with œdema, ascites, and transudation into the pleural cavities or pericardium.

Other situations in which lympho-sarcomata originate are the retroperitoneal glands, the

MALARIA

kidneys, and occasionally the deep lymphatic glands of the pelvis.

It has already been noted that the term lympho-sarcoma is used with extreme vagueness by many surgeons to denote any rounded tumour of the sarcomatous type and there is no doubt that in the comparatively recent past, enlarged glands which to-day are recognized at once as lymphadenomatous have been described as belonging to this group. The diagnosis can only be made by careful microscopic examination of the tumour.

Prognosis and treatment are alike quite hopeless.

HUGH THURSFIELD.

LYMPHURIA (see FILARIASIS).

MACROCEPHALY (see SKULL, ABNORMALITIES IN SHAPE OF).

MADURA FOOT (*syn.* Mycetoma).—A disease, especially common in India and Northern Africa, caused by infection with any of several forms of streptothrix. Though the foot is the part most commonly attacked, the hand and other parts are not exempt. The fungus (PLATE 28, Fig. 9, Vol. III, facing p. 138) is probably parasitic on various cereals, and gains access to the naked feet of agricultural labourers in the tropics through some small abrasion in the skin.

Pathology.—The changes produced in the tissues are very similar to those caused by the streptothrix of actinomycosis. The fungus proliferates in the connective tissues of the foot, and masses of mycelium form, and lead to softening in their immediate vicinity, and a defensive induration a little more remotely. The soft material comes to the surface and sinuses are formed through which are discharged numerous granules composed of the mycelium. These granules may be black, yellow, white, or red in colour; in the first variety the whole foot may have a dark appearance. The tarsal bones may be eroded or softened.

Symptoms.—A firm, painless nodule develops in the sole of the foot. Later the concavity of the sole is filled in and may become convex as the result of the extensive tissue

reaction. The whole contour of the foot is altered. Sinuses form and discharge an oily-looking fluid containing the granules. Pain may now be complained of when any pressure is put on the foot. The limb wastes and becomes useless, and the patient may be seriously weakened by the constant discharge.

Diagnosis.—In the early stages diagnosis may be impossible, but a firm, painless nodule in the sole of the foot of a native labourer should arouse suspicion. The diagnosis is easily made when the granules can be obtained in the discharge, and still more so in the later stages.

Treatment.—In the early stages it is possible to excise the initial nodule, but cases are rarely diagnosed sufficiently early. In late cases amputation of the limb well above the disease is called for. If operation be not undertaken, some benefit may accrue from the administration of large doses of potassium iodide, or from inoculation with a suitable vaccine prepared from the infecting organism.

ZACHARY COPE.

MALARIA (*syn.* Ague; Paludism; Inter-mittent Fever; Remittent Fever; Marsh Fever; Jungle Fever; Climatic Fever; Coast Fever).—The term malaria covers three specific fevers of protozoal origin, caused respectively by *Plasmodium malariae*, *P. vivax*, and *P. falciparum*, the infection being transmitted by anopheline mosquitoes. The fevers are charac-

terized clinically by periodicity of the attacks, enlargement of the spleen, and quick response to quinine-therapy.

History.—In the old Indian system of medicine, "Charaka-Samhita," there are references to fevers spread by mosquitoes, and it has been suggested that these statements relate to malaria. Hippocrates clearly described quotidian, tertian, and quartan types of fever. In 116 B.C., Varro emphasized the relationship between malaria and marshes. Later, Celsus differentiated two types of tertian fever, the benign and the malignant. The association of malaria with stagnant water was generally recognized during the Middle Ages. In the seventeenth century the therapeutic properties of cinchona bark were discovered. In 1638, the Countess Cinchon, the wife of the fourth Count, Spanish Viceroy of Peru, contracted tertian malarial fever, and no treatment was of any avail until the infusion of the bark of the tree known to the Indians as Quina-quina was administered. The Countess on her return to Europe brought home a large supply of the bark, which she distributed to persons suffering from fever on her estates near Madrid. Hence the bark came to be known as "pulvis comitisse." Linnæus called the tree after the Countess, giving it the name of "*Cinchona officinalis*." In 1820, Pelletier and Caventon prepared an alkaloid, quinine, from cinchona bark.

As regards the actual cause of the disease, for centuries it was believed to be minute forms of animal life arising in the exhalations from decaying vegetable matter in marshy land. In 1847, Mackel noted the presence of blood pigment in the organs of people dying from malaria. In 1880, Laveran in Algeria discovered the causative parasites while carrying out an investigation on the origin of the pigment. He called the parasite *Oscillaria malariae*. At first his great discovery did not attract much attention, many authorities believing in a bacterial origin of the disease. In 1886, Golgi demonstrated the life-cycle of the parasite in man. The suggestion was made by King, in 1883, that mosquitoes might be the carriers of the infection, which, as already noted, was the belief of the oldest observers. Manson, in 1894, definitely formulated the hypothesis of the mosquito transmission of malarial fever, basing it upon the observations he had made of the flagellation of the male gamete, which does not take place until several minutes after the removal of the blood from

the general circulation. In 1898, Ronald Ross discovered that the malarial parasites could grow in the stomach of a mosquito, and eventually found the whole cycle of the parasite of bird malaria in that insect; and this was subsequently completed, as regards the human parasite, by Grassi, Marchiafava and Bignami. Manson succeeded in causing typical attacks of malaria, in persons who had never left Great Britain, by the bites of anopheline mosquitoes infected with parasites in Italy.

Geographical distribution.—Malaria is widely distributed all over the tropics and subtropics and in many parts of the temperate zones; in Europe it is extremely common in the Balkans and the south of Italy; in France it is found in the southern and western provinces; in Switzerland, in the Canton of Tessin; in Germany, in the lowlands watered by the tributaries of the Danube. Certain parts of Great Britain were very malarious in the Middle Ages, especially some districts bordering the lower Thames and large parts of Cambridgeshire and Lincolnshire. At one time it was generally believed that indigenous malaria had completely disappeared from this country, but recent researches have shown that there have always been some small endemic foci.

Pathology and morbid anatomy.—The pathological features of the malady are due principally to the destruction of enormous numbers of erythrocytes, and quite probably also to the presence of toxins, hæmolyisins and endotheliolysins produced by the parasites.

Cases due to benign tertian and quartan malaria rarely end fatally, and our knowledge, therefore, of the morbid anatomy of the malady is based almost entirely on cases of pernicious malaria (subtertian). The spleen is usually enlarged, hard, and of a dark colour, but in acute cases it may be soft and diffuent. The liver is in most cases enlarged, and varies from a dark brown to a slate hue. Microscopically, the endothelial and Küpfer cells show a large amount of black pigment (hæmozoin), while the parenchymatous cells contain no such pigment, but often yellow granules (hæmosiderin). Hæmozoin, although containing iron, does not give the iron reaction, and is soluble in alkalis but not in alcohol. Hæmosiderin, on the other hand, is soluble in alcohol but not in alkalis.

The bone-marrow is very dark, owing to the presence of a large amount of pigment. In pernicious cerebral cases the brain has a leaden

colour, and microscopical examination of sections of it will often show the capillaries to be blocked with degenerated red cells, pigment, and parasites. The lungs may be normal, anæmic or hyperæmic, or congested. The suprarenal capsules may be hyperæmic; the kidneys generally appear to be normal, but occasionally they may be congested or of a brownish colour.

Etiology. The parasites in man. (PLATE 18.) *Benign tertian*.—The parasite of benign tertian (*P. vivax*), as seen in the red corpuscles, appears at first as a roundish, unpigmented body, about $2\ \mu$ in diameter. A few hours later it increases in shape and becomes ring-like; then a few granules of pigment appear and the parasite shows active amoeboid movements. It gradually enlarges until at the end of 48 hours it occupies almost the whole of the red corpuscle; between 40 and 48 hours, many of the parasites undergo segmentation into 15 or 20 sporiform bodies, which frequently show a radial arrangement. A certain number, however, do not undergo any process of segmentation, and these represent the sexually differentiated forms or gametocytes.

Quartan.—The parasite of the quartan fever (*P. malaria*) at first is very similar to the tertian parasite in appearance, but later the granules of pigment are larger and darker, and their movement is not so marked. On the third day segmentation takes place, the pigment collecting toward the central portion of the parasite, which finally breaks up into 6-12 segments. Some of the full-grown parasites, however, as is the case with *P. vivax*, do not segmentate (gametocytes).

Malignant tertian (or subtertian).—*P. falciparum* appears at first as a very small hyaline body, smaller than the tertian or quartan parasites, which quickly forms a ring. The pigment is present in very small amount, or altogether absent at first; the later stages of development are not ordinarily seen in the peripheral blood, these forms being found in the blood of some internal organs, principally the bone-marrow and the spleen. After about a week, large crescentic bodies appear, containing a great deal of pigment in the centre. These are the so-called "crescents," which are characteristic of subtertian fever. They do not sporulate; they represent sexually different forms (gametocytes), and are incapable of further development in the human host, but in the stomach of the mosquito the male elements (microgametocytes) produce a number of motile flagella (microgametes), which become detached and fecundate the female bodies (macrogametes).

The parasite in the mosquito.—When an anopheline mosquito bites a human being in whose blood gametocytes are present, fecundation of the female element takes place in the insect's stomach. The fecundated female element penetrates the walls

of the mosquito's stomach and a definite cycle of development takes place in the muscular coat, roundish, refractive bodies that contain pigment appearing, and gradually enlarging until after about a week they reach a diameter of 60-70 μ . These large bodies (oöcysts or zygotes) then burst, and an enormous number of spindle-shaped bodies or sporozooids are set free in the body cavity of the mosquito; they accumulate in the salivary glands of the insect, and when it bites they escape into the ducts and are inoculated.

The anopheline mosquito (PLATE 19, Figs. 1, 2, 3).—Only mosquitoes of this family are capable of transmitting malaria. This is not the place to enter into zoological details, but for practical purposes, when it is desired to determine a mosquito the first step to take is to see whether the insect is a male or female, as the male does not bite human beings. The antennæ of the male are very plumose, while those of the female show only a few hairs, and if the antennæ have been broken off one should look for the claspers at the end of the abdomen. The female Anophelinae differ from the common female Culicinae in the following characteristics: The wings are generally spotted (dappled), the palpi are as long as the proboscis, or longer; when the insect is in a resting position the abdomen, head and proboscis are in a straight line, and it does not show the hunchback appearance of the Culicinae. The scutellum of Anopheles is simple, while that of Culex is trilobed. The eggs of Anophelinae are generally collected in star or ribbon-like arrangement, while the eggs of the Culicinae are collected in raft-like formations; the anopheline larvæ are asiphonate and take a horizontal position, while the Culicinae larvæ are siphonated and take a 45°-angle position. The male Anophelinae feed on the juices of flowers and fruits.

Symptomatology. Benign tertian.—The incubation period is generally 8-10 days; in experimental cases, when healthy men have been bitten by infected mosquitoes, it varies from 7 to 25 days.

The febrile attack.—The patient for a day or two may be feeling out of sorts and may complain of rheumatic-like pains together with a feeling of lassitude, but at times there are no prodromata. He feels suddenly very cold, and actual shivering may set in; the teeth may chatter, the lips become blue, the legs and arms are cold, and gooseskin may be present. This "cold stage" lasts 10-30 minutes. The chills then diminish, sensations of warmth appear, at first pleasurable, but they soon become intense and the patient feels burning hot; the skin is hot, dry and flushed, the conjunctivæ injected, the pulse full and occasionally dirotic; at times a slight cough may be present, and there may be vomiting and

PLATE 18.—MALARIAL PARASITES OF MAN.

(Description of Plate drawn by John Gordon Thomson, M.A., M.B., Ch.B.)

A. BENIGN TERTIAN PARASITE (*Plasmodium vivax*).

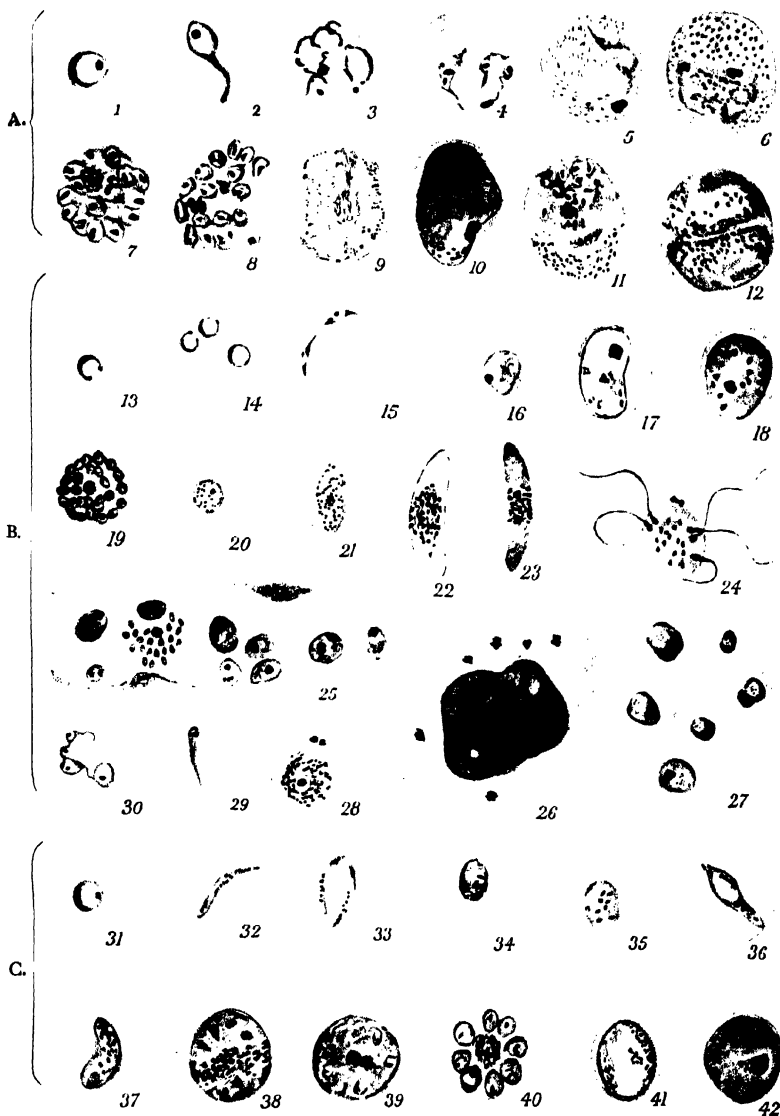
1. Trophozoite. Signet-ring form.
2. Trophozoite showing amoeboid characteristics.
- 3 and 4. Corpuscles containing several young parasites showing amoeboid characters. The parasites also show the so-called "tenué" phase.
5. Shows distinct enlargement of the containing corpuscle, and the presence of Schüffner's dots within its cytoplasm. This enlargement of the corpuscle and these dots are very characteristic of *P. vivax*. The parasite is actively amoeboid.
6. Trophozoite about half-grown showing the chromatin divided into three portions. Pigment is scattered throughout the cytoplasm. The corpuscle is enlarged, and shows Schüffner's dots.
7. Schizont containing 16 merozoites. The pigment has collected.
8. Schizont in process of disruption.
9. Male gametocyte. The pigment, which is in fine granules, is scattered. The cytoplasm stains faintly, and the chromatin, while more or less centrally placed, is somewhat scattered.
10. Female gametocyte. The pigment is in coarser granules. The cytoplasm stains more deeply, and the chromatin is compactly grouped on the margin.
11. Gametocyte and schizont in the same corpuscle. This combination gave rise to Schaudinn's erroneous theory of parthenogenesis.
12. Two gametocytes in the same corpuscle.
22. Male crescent or gametocyte. Pigment in finer granules and more scattered, cytoplasm a fainter blue, and the chromatin more scattered than in the female.
23. Female crescent or gametocyte. Pigment in coarser granules tending to be grouped nearer the chromatin, chromatin more compact, cytoplasm stains a deeper blue.
24. Extragellation of a male gametocyte. This occurs when blood is drawn. The crescent becomes rounded, and the chromatin undergoes reduction (maturation) division with the extrusion of two polar bodies. Five microgametes have been formed.
25. Capillary from a smear of the brain, showing the lumen packed with parasites. One of these is a mature schizont showing the merozoites.
26. Endothelial cell from the peripheral blood of a comatose case. It contains pigment which it has phagocytosed.
27. Clump of red blood-corpuscles from the peripheral blood of a comatose case. This illustrates the tendency of corpuscles containing the malignant tertian parasite to clump together.
28. Female gametocyte undergoing reduction or maturation division with the extrusion of two polar bodies.
29. From a comatose case showing an unusual band-shaped trophozoite.
30. Multiple infection of a corpuscle with young trophozoites, the so-called "tenué" phase.

C. QUARTAN PARASITE (*Plasmodium malariae*).

B. SUBTERTIAN PARASITE (*Laverania malariae*, *Plasmodium falciparum*).

13. Trophozoite (ring form) in the peripheral blood. Shows the chromatin in two masses.
14. Multiple infection of a corpuscle with trophozoites (rings). From a film of the peripheral blood.
15. Trophozoites on the margin of a corpuscle. Peripheral blood.
16. Trophozoite from a capillary of one of the internal organs, showing pigment already collected into a dense mass.
17. From smear of one of the internal organs. Shows developing schizont, in which the chromatin has broken up into several masses.
18. Developing schizont more grown than No. 17.
19. Schizont containing 27 merozoites. The mature schizont fills roughly two-thirds of the corpuscle. Found in the capillaries of the internal organs.
20. Developing crescent from a smear of bone-marrow. The pigment is scattered.
21. Developing crescent becoming elongated and oval. From a smear of the bone-marrow. The pigment is scattered.
31. Trophozoite (ring stage).
32. Trophozoite. Illustrates the band shape of the parasite. Pigment has appeared and is scattered throughout the cytoplasm.
33. Band-shaped trophozoites.
34. Trophozoite. Compact parasite showing chromatin just beginning to divide. The pigment is scattered throughout the body.
35. Trophozoite.
- 36, 37. Show the characteristic band shape of the trophozoite.
38. Developing schizont. The chromatin has divided into 9 masses, and the pigment is collected into a band-shaped mass.
39. Developing schizont. The merozoites have not yet completely separated off.
40. Schizont (rosette) containing 8 merozoites. The pigment is collected towards the centre.
41. Male gametocyte. Smaller than the female. The cytoplasm stains more faintly, the pigment is in finer granules, and the chromatin more diffuse.
42. Female gametocyte. The cytoplasm stains more deeply, the chromatin is more compact, and the pigment, in addition to being in larger masses, is more plentiful.

All the preparations were stained with Giemsa, and drawn at a magnification of approximately 2,200 diameters.



JOHN GORDON THOMSON, del.

PLATE 18.—MALARIA PARASITES OF MAN.



rarely, diarrhoea. The spleen is often palpable and tender. The temperature begins to rise before the chill, and continues to ascend during the cold stage, reaching the maximum in the hot stage. It may be anything from 102° to 108° F., but is generally between 104° and 105°. The warm stage may last four or five hours, and is followed by the sweating stage. Profuse perspiration breaks out all over the body, and during this stage the pulse, respiration, and temperature may fall to normal in about four hours, after which the patient very frequently goes to sleep. The pyrexial attack lasts altogether about 10 or 12 hours. In most cases it begins in the morning, but it may take place at any time of the day. After the temperature has fallen to normal the patient as a rule feels quite well. At the end of 48 hours however, another pyrexial attack generally develops with all the symptoms shown by the first. Benign tertian malaria, left to itself, tends in most cases to spontaneous cure after a variable number of febrile attacks.

Double tertian fever.—When tertian parasites mature on two separate days, fever is produced every day, and is therefore quotidian in type and is called double tertian (*tertiana duplex*).

Quartan fever.—The incubation period is generally stated to be about 10 days, but may be much longer.

The febrile attack.—Some few hours before the pyrexial attack the patient may complain of giddiness, weakness, malaise, headache and nausea, and perhaps of muscular pains all over the body. In a short time the definite attack begins, which again may be divided into three stages: the cold, hot, and sweating. At first the patient feels very cold, and shivering sets in, the rigors being often very emphatic. The cold stage lasts 15–30 minutes, and is followed by the hot stage, when he feels warmer and more comfortable, but gradually the sensation of heat increases, the skin becomes very hot and dry, and the conjunctivæ injected; the pulse is frequent and the respiration increased. This stage lasts about 3–4 hours, and then the sweating stage begins. Sweating is profuse, and brings great relief, the temperature subsiding very rapidly and the pulse-rate declining. After the fall of temperature the patient often feels quite well, though sometimes weak, but on the fourth day another pyrexial attack develops in every way similar to the first.

Double quartan fever.—There is an attack of fever on two successive days, and an apyrexial

interval of 24 hours. This is due to two broods of parasites having been inoculated on separate days.

Triple quartan fever.—This is caused by three broods of quartan parasites coming to maturity on three successive days; the fever has a quotidian periodicity.

Malignant tertian (subtertian).—The incubation period is 9–10 days, and prodromal symptoms resembling those of benign tertian may be present. The cold stage, when present, is very similar to the cold stage in benign tertian, but in certain cases it is entirely absent and the attack may begin with the warm stage. The skin is flushed and dry, and occasionally subicteric; the conjunctivæ are injected. The sweating stage is never absent and is generally pronounced. The spleen is tender and palpable, and so is often the liver. The duration of the pyrexial attack is about 24 hours. The pyrexial periodicity is fairly often a tertian one, there being an apyrexial interval of 24 hours, but this interval may be much shorter (12 or 10 hours).

Double subtertian fever.—This is a quotidian fever brought on by two broods of *P. falciparum*. As a rule the cold stage is very little marked. The temperature rises rapidly, the fastigium is very short, and the fall is to below normal. The whole attack lasts 6–12 hours.

Atypical forms of malaria.—In certain parts of the tropics and of Southern Europe atypical cases of malaria, generally due to the parasite of malignant tertian, are commonly met with, and the patient may present symptoms pointing to widely different pathological conditions; indeed, there are very few internal diseases which malaria may not simulate, and it may even give rise to syndromes closely resembling surgical conditions such as appendicitis. The symptoms may be extremely severe, and the prognosis is often bad (pernicious malaria). These forms may be grouped as follows:

NERVOUS SYSTEM.—Nervous syndromes are comparatively common.

Comatose form.—The patient very quickly becomes comatose, and cannot be roused to answer questions; he will only moan. Respiration may be stertorous or sometimes quiet. The pupils are often contracted; there may be high fever, or the temperature may be normal or subnormal, and in the latter cases the diagnosis of opium poisoning has occasionally been made. The comatose type of malaria is of great practical interest, as it may develop

MALARIA

suddenly in subjects with latent malaria many years after leaving the tropics.

Delirious form.—In this type delirium is the most prominent feature, so that the cases have occasionally been mistaken for alcoholic delirium tremens.

Meningitic form.—The patient complains of very severe headache; there are retraction of the head and rigidity of the neck, vomiting, convulsions and hyperæsthesia. All the symptoms disappear on administering quinine in full doses.

Mental form.—Malaria may occasionally simulate mental diseases, and cases of apparent mania and melancholia are on record.

Hemiplegic form.—During or after a severe pyrexial attack the patient suddenly develops typical hemiplegia. Cases of monoplegia also occur.

Cerebellar form.—The patient may have very low fever or none at all; he complains of severe headache and almost complete loss of vision; his walking is like that of a drunken man. Vomiting is frequent. Quinine-therapy cures the condition.

Polyncuritic form.—In some cases there is œdema; the gait is stepping and the knee-jerks are lost, the clinical picture closely resembling wet beriberi. In other cases there is no œdema, and the patient may present signs of severe muscular atrophy.

Korsakoff-like form.—Symptoms of polyneuritis develop and are accompanied by mental symptoms, among which is loss of memory for recent events.

DIGESTIVE SYSTEM.—The *gastric form* is characterized by acute or chronic dyspeptic symptoms showing often a certain periodicity; for instance, the attack of indigestion in certain cases comes on every third day. There may be no fever, and the spleen and liver may not be enlarged. Dietetic treatment has no influence whatever, and the symptoms disappear only on the administration of quinine.

Pseudo-dysenteric form.—Two varieties of this form may be distinguished: in one the stools contain blood and mucus, in the other they contain blood but no pus and very little or no mucus. It should not be forgotten, of course, before ascribing the dysenteric condition to malaria, that cases of mixed infection, malaria and bacterial or amœbic dysentery, are quite common. In malarial dysentery quinine acts very rapidly.

Choleraic form.—The patient is taken ill suddenly, very often at night, with profuse

diarrhœa, and there may be complete absence of fever. He feels very cold, the skin is clammy, the cheeks are sunken; the stools may be cholera-like with the familiar rice-water appearance. Quinine cures the condition very rapidly.

Cholecystic form.—These cases closely resemble acute cholecystitis due to calculi; there are severe pain shooting towards the right shoulder, tenderness in the region of the gall-bladder, severe vomiting, and at times a slight jaundice. The spleen is often slightly palpable, and the blood-examination reveals malarial parasites. Under quinine-therapy all the symptoms disappear very rapidly.

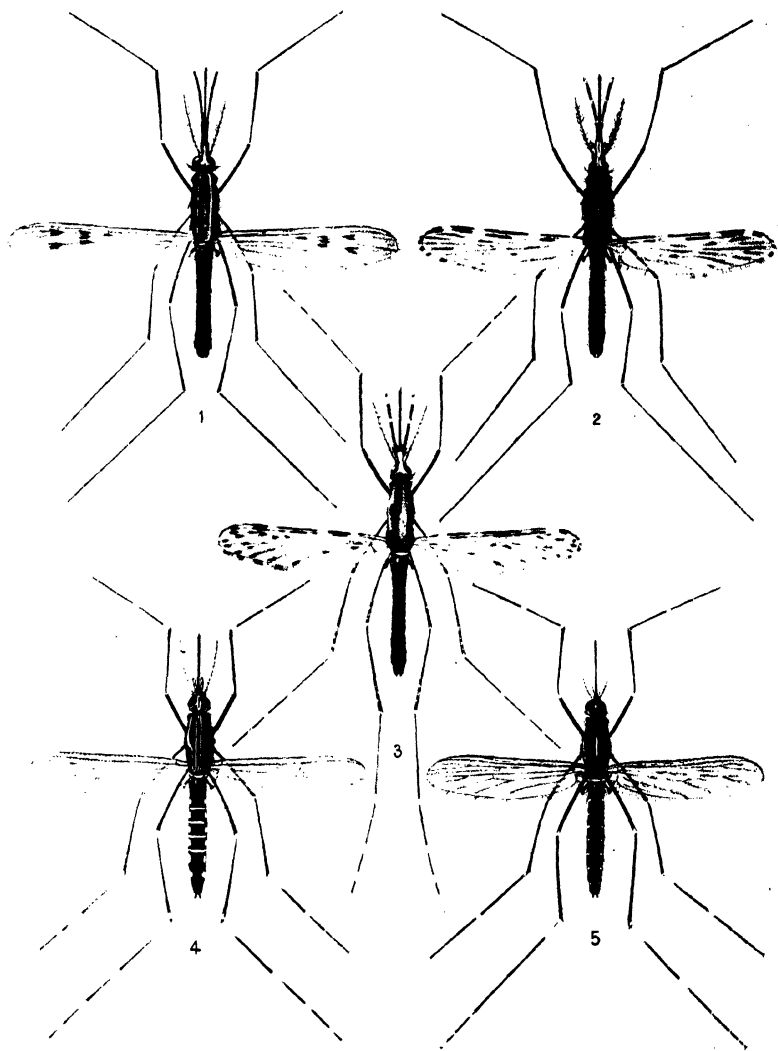
Appendicular form.—The patient is taken ill with violent pain in the appendicular region, and there may be severe vomiting and fever; on palpation there is tenderness, often with pronounced rigidity of the right lower abdomen. Quinine is rapidly efficacious.

RESPIRATORY AND CIRCULATORY SYSTEMS.—Dry bronchitis of malarial origin is not very rare; and cases of consolidation of the lung, due to the same cause, are also observed, closely resembling lobar pneumonia. As regards the circulatory system, symptoms of angina pectoris and various types of tachycardia and arrhythmia have been recorded.

UROGENITAL SYSTEM.—Malarial hæmoglobinuria, or malarial blackwater, is a serious condition not rarely met with in tropical and subtropical countries; it should be distinguished from quinine hæmoglobinuria and from true blackwater fever. The patient has an attack of very high fever, shivering, severe vomiting, great prostration and rapid anæmia, the urine showing a characteristic stout-like colour.

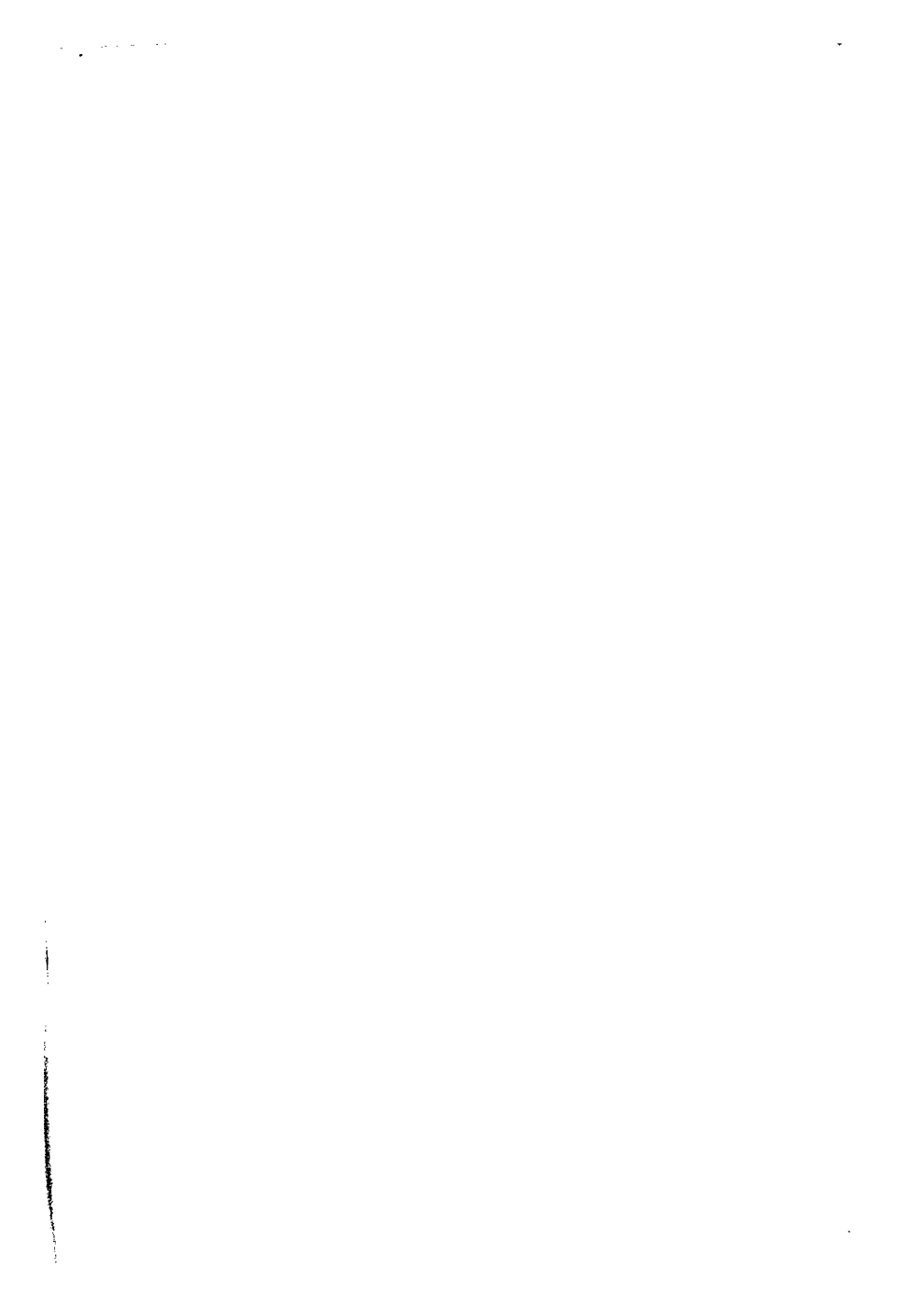
Cases of malarial nephritis have been recorded but are rare. Neuralgia of the testicles and neuralgia of the ovaries of malarious origin are occasionally met with. A malarial orchitis has been described.

THE SKIN.—Various skin eruptions may be seen in malaria, the most frequent being herpes labialis. Occasionally a diffused simple erythema appears, or an urticarial rash may develop. Cases of purpura have also been observed. In chronic cases the skin is often earthy in colour, or it may be pale yellow or ashy grey. Patches of hyperpigmentation (chloasma malaricum) are common, and occasionally a diffuse dark pigmentation is seen in conjunction with great loss of flesh and severe



1, *Anopheles maculipennis*, $\times 5$. 2, *A. funestus*, $\times 10$. 3, *A. maculatus*, $\times 5$. 4, *Stegomyia fasciata*, $\times 7$. 5, *S. pseudoscutellaris*, $\times 7$. (All females.)

PLATE 19.—PARASITE-BEARING MOSQUITOES.



asthenia, the condition resembling very closely Addison's disease.

Diagnosis.—The classical signs on which to base a diagnosis of malaria are: (1) The periodicity of the fever. (2) Enlargement and induration of the spleen. (3) Specific action of quinine on the temperature. (4) Most important of all, the finding of malarial parasites in the blood.

The practitioner should always bear in mind that in a number of cases some of these features may be absent, and that a negative blood-examination is of no importance whatever unless the search for parasites is repeated very many times. While laboratory examinations are of great importance—it is only by this means, indeed, that a positive diagnosis can be made with absolute certainty—due account should also be taken of the clinical examination. The importance of simple inspection and of simple palpation can hardly be exaggerated; a slight abnormality of the colour and pigmentation of the patient's skin, overlooked by the inexperienced, may suggest to the expert tropical clinician the true diagnosis in an obscure case, and acting on it he may perhaps save the patient's life; in many difficult cases the faintest touch of the tip of the spleen, impalpable to less trained hands, may make the diagnosis clear.

Prognosis.—Provided that an appropriate quinine treatment be instituted, the prognosis is favourable in the great majority of cases. This, however, is subject to certain qualifications. (1) Pernicious cases may be attended by a high mortality in spite of energetic quinine treatment. (2) In a large number of cases, even of the usual benign type, the cure which is brought about by an appropriate treatment is merely clinical, i.e. all the symptoms of the disease disappear and the patient feels well, but complete sterilization as regards the parasite is not achieved. The affection remains dormant for months and years, and anything that lowers resisting power, such as a chill or traumatism, may bring about a recurrence of the acute symptoms in patients who have been in England for ten or fifteen years after leaving the tropics. As regards traumatism, the simplest operative act may reawaken a very old dormant affection; the patient, shortly after the operation, may have a rigor, the temperature rapidly rises to 104° or 105° F., and very severe vomiting sets in—symptoms which may give great anxiety to the surgeon, who naturally may be very far from thinking of malaria.

Treatment.—The treatment of malaria may be condensed into one word, **quinine**. The drug should be given in full doses, and its use should be continued in decreasing doses for at least three months after the last attack of fever.

Dosage.—During the acute and subacute stage the drug should be given in 10-gr. doses three or four times a day in adults. In children, too, the amount should not be small; the following quantities are suitable:

Under 1 year	..	1 gr.	three to six times daily.
1 to 3 years	..	2 gr.	" "
3 " 10 "	..	3 gr.	" "
10 " 15 "	..	5 gr.	" "

Modes of administration.—Quinine should be given by the mouth in ordinary cases, by intramuscular injection in severe cases or when vomiting is present, and intramuscularly and intravenously in pernicious cases. When given *by the mouth*, the old sulphate or bisulphate is the best preparation, and the following mixture will be found useful for routine administration:

℞	Quin. sulph.	10 gr.
	Acid sulph. dil.	℥x.
	Syr. aurant.	℥i.
	Aq. or aq. chlorof.	ad ℥i.
	T.d.s.	

When the mixture induces severe tinnitus aurium, 5 or 10 gr. of potassium bromide may be added to each dose; in cases in which the heart is weak, 2-5 min. of tr. digitalis may be added.

Tablets and pills, each containing 5 gr. of quinine bisulphate or bihydrochloride, are very convenient for patients who object to the bitter taste of the mixture; they are not, however, so efficacious.

Quinidine and cinchonidine are very useful in some chronic cases. In such cases I generally give quinidine sulphate in 10-gr. doses thrice daily.

The *intramuscular administration* of the drug is now deprecated by several authorities owing to the accidents which have been recorded, but in my experience it is a very valuable method, and provided that the injections are carried out with proper precautions no accidents need be feared. Special attention should be paid to these points: (1) The solution should be prepared accurately and properly sterilized, and should be non-irritant. The simplest way is to use ampoules, each containing 15 gr. of the bihydrochloride, obtained from a reputable firm. (2) The skin should be thoroughly disinfected, the application of tincture of iodine being the simplest procedure. (3) The injec-

tions should be made deep into the muscular masses of the gluteal regions, not merely subcutaneously. (4) Care should be taken never to give the injection in a spot which is infiltrated from a previous one. (5) The positions of main vessels and nerves should be avoided.

Time of administration.—Some years ago great stress was laid on this consideration. Most authorities, and especially Koch, insisted on the so-called "rational method," according to which quinine should not be given while the temperature is rising. In my experience, however, the safest rule is to give quinine directly a probable diagnosis of malaria has been made. The practitioner should never forget that what seems to be a mild attack of fever may occasionally turn into a pernicious one with sinister abruptness.

Noxious effects of quinine. Some subjects have a serious idiosyncrasy to the drug; but such cases are comparatively rare, and generally the symptoms due to quinine are merely buzzing of the ears, slight temporary deafness, and occasionally slight tremor of the hands. The addition of 5–10 gr. of potassium or sodium bromide, per ounce, to the quinine mixture mentioned above is sometimes very useful in preventing these symptoms. A distressing condition which may be caused by quinine is partial amaurosis; this, however, is fortunately very rare, and usually is only temporary. It is important to differentiate quinine amaurosis from malarial amaurosis. In the former the pupils are generally widely dilated, and do not react to light; in the latter, reaction to light is present.

Nursing, diet, and symptomatic treatment.—During the pyrexial attacks the patient should be kept completely at rest in bed, on fluid or soft diet. In the apyrexial periods he may be allowed up and may have plain solid though light diet. As regards symptomatic treatment, constipation must be guarded against and should be relieved promptly by a dose of calomel, 1–3 gr., followed by a saline. Vomiting, which is often very distressing, can be relieved by sips of iced soda-water or champagne. As already stated, when the vomiting is severe quinine should be given not by the mouth but by intramuscular injection. Splenic pain, which is often due to perisplentitis, may be relieved by hot fomentations or the application of belladonna ointment. Hyperpyrexia must be treated by cold sponging or cold packs; algidity by saline injections and warm applications to the body. When hæmoglobinuria is

present, calcium lactate should be administered and stimulants given.

Other drugs. An enormous number of drugs have been tried, but their efficacy is very slight, and none of them has a definite specific action on the malarial parasite. Among them are methylene-blue, arsenic, tartar emetic and phosphorus. Methylene-blue is occasionally useful in cases of idiosyncrasy to quinine; it may be given in cachets each containing 2 gr., one cachet three times daily. It must be admitted, however, that as a rule the results are not satisfactory. Ordinary preparations of arsenic are of some use in chronic cases when given in conjunction with quinine. Salvarsan and neosalvarsan have been very highly praised in the treatment of malaria by some observers, but in my experience their action on the malarial parasite is very slight. Tartar emetic has no specific action on malaria, nor has phosphorus, but both drugs occasionally seem to reinforce the action of quinine. I have used phosphorus by subcutaneous injection, employing the ordinary phosphorated oil of the B.P., 1 or 2 min. diluted with 20 min. of sterile olive oil.

Prophylaxis.—This is based principally on anti-mosquito measures and secondarily on the preventive administration of quinine. In practice, the most useful anti-mosquito measure is the destruction of the larvæ by oiling all pools and ponds near houses, camps, hospitals, etc., with crude petroleum or sanitasokol or izo-izal. Daily inspection of compounds should be carried out, special attention being paid to absence of stagnation of water in gutters and to the careful collection and disposal of household utensils likely to harbour mosquito larvæ, as discarded tins, etc. Anti-mosquito protection by means of mosquito nets, mosquito masks (Simpson's mask), etc., should also be carried out. Chemical preparations to keep off mosquitoes are occasionally advantageous; plain citronella oil may be used, or a menthol powder such as menthol 3–5 gr., zinc oxide to 1 oz.

The prophylactic administration of quinine does not give results comparable with those obtained by anti-mosquito measures efficiently carried out, but is useful; I have always found it very advantageous to combine the two methods. There are various methods of giving quinine prophylactically; I generally recommend taking 5 gr. every day in the week and a double dose on Sundays, or 10 gr. on Saturdays and Sundays. In very malarious districts the dosage should be doubled.

ALDO CASTELLANI.

MALINGERING

MALIGNANT ENDOCARDITIS (see ENDOCARDITIS, ACUTE).

MALIGNANT PUSTULE (see ANTHRAX).

MALINGERING.—The term malingering was originally applied to soldiers and sailors who pretended illness in order to escape duty, and until recent years it was employed practically only in military medicine. But when it became possible to obtain compensation for injuries sustained in railway travelling and other circumstances, and when the Workmen's Compensation Act entitled workers to receive monetary payment for incapacity due to accidents which occurred in the course of their work, it was natural that dishonest persons should feign disabilities. The National Health Insurance Act, under which the insured obtain sick benefit while unfit for work, has given further opportunities to the dishonest to malingering, and may even induce them to do so, particularly if by further insurance the benefits they receive approximate to the working wage.

But malingering, in the sense of simulation of an illness or disability for which there is no basis, is rare, certainly much rarer than it is usually assumed to be. On the other hand, it is not uncommon to find the symptoms due to an illness or accident exaggerated or protracted when the patient can thereby obtain some direct benefit; after a blow on his back a man may exaggerate the pain and the disability, or may continue to complain of them when an actual cause for them no longer exists. The French, who recognized the frequency of these types during the late War, applied to them the titles *exagérateurs* and *persévérateurs*. Finally, a man may either honestly or dishonestly attribute to an accident or injury disabilities which are quite independent of them; if he does so dishonestly, for the sake of personal gain, he must be classed as a malingerer.

Malingering may be consequently defined as a deliberate and designed feigning of disease or disability which is non-existent; or a conscious exaggeration or protraction of symptoms due originally to some physical cause; or a false and dishonest imputation, for the purpose of self-benefit, of symptoms to an accident or injury which had no causal relation to them.

Attempts have been made to subdivide malingering into types or varieties, but so many cases are intermediate between the types that

have been differentiated, or possess features common to more than one type, that we cannot formulate a useful or accurate classification. On the one hand there is the fraudulent swindler who, without any reasonable excuse, seeks to profit from an accident by simulating a disability, or by attributing to it some condition that pre-existed, or that he knows is not due to it; and on the other, the person with a justifiable claim who, without a definitely dishonest intention, exaggerates his symptoms lest he should not receive the compensation to which he believes he is rightly entitled. Most malingerers come between the extremes. The most common type may be illustrated by a man who, as a result of an injury, has acquired a trivial or temporary disability, which he exaggerates while his symptoms persist, or who continues to complain of them when they no longer exist, and on this basis claims compensation owing to an incapacity for work. After a blow on the back that has not produced any permanent injury, he may complain for months of pain, or of inability to straighten himself up; or after a trivial accident to his leg he may pretend to be unable to move it, or to walk properly on it. He is consciously exaggerating and protracting the temporary symptoms that were due to the accident. Frequently, however, the claim is not so directly dishonest; during the time he is really incapacitated the compensation that is assured to him relieves him of worry as to his immediate future, and necessity is not present as an incentive to pull himself together and do as much as he can. Then sympathetic friends, and not infrequently legal advisers, discuss with him the possible ill effects of the accident, and suggest future dangers so strongly that he begins to brood on them, becomes introspective, interprets every abnormal sensation as a sign of some serious disease, and his attention becomes so absorbed in the accident and its effects that his whole life seems to him to turn around it. This man is now in a state of "traumatic neurasthenia," and his claim for compensation, as far as this condition goes, is honest and justifiable; but in his statement for claims and during expert examination there is often a deliberate exaggeration of his symptoms, and frequently a simulation of symptoms, suggested perhaps by a leading question or by an unguarded word, and to this extent he is a malingerer too. This hypothetical but typical instance illustrates the greatest of the difficulties we meet with in considering malin-

gering—the difficulty of distinguishing it and, when it exists, of separating it from the traumatic neuroses, and especially from hysteria.

Diagnosis of malingering.—The medical man who has frequently to consider the question of malingering requires a thorough knowledge of general medicine and surgery, and of their special branches, or he must at least know when it is necessary to call in expert help. And knowledge of men, and especially of men of the class with which he has to deal, is equally essential. His first duty must be to exclude the existence of any real disease to which the symptoms may be due, or to assure himself that the disabilities which the patient asserts do not depend on any disease that may be present; then he has to consider the possibility of hysteria or traumatic neurasthenia; and finally, if he is satisfied that the patient's claims are fictitious, he must endeavour to furnish demonstrative evidence of malingering.

1. **Exclusion of organic disease.**—This should be assured by a thorough clinical examination, directed particularly to those organs and parts to which the symptoms are referred. Use should be made, when necessary, of the pathologist and of all accessory means of diagnosis that may be of service, especially of radiography, as, in addition to furnishing valuable positive or negative information, this places the malingerer at a disadvantage, as he knows not what it may or can reveal. It is impossible, in an article of this length, to deal with even the more important problems that arise in excluding organic disease of the various viscera, but as the majority of malingerers base their claims on pain, or on paralysis, or other such nervous disability, we must consider shortly the features that characterize injuries and diseases of the nervous system.

Palsies due to *affections of the peripheral nerves* can be easily recognized, as those muscles only that are innervated by the injured nerves are weak, while in hysteria and in the paralysis simulated by the malingerer it is invariably movements that require the co-operation of several muscles that are affected. Further, when a lesion of a nerve produces weakness it also entails wasting and electrical changes in the paretic muscles. Weakness due to *lesions of the central nervous system* is distinguished by its distribution, by its nature, and by the associated reflex and sensory alterations. That due to injury of the motor cerebral cortex, or of the upper motor neurones that take origin in it, almost invariably affects

all movements of a limb, but unequally, the distal segments being, as a rule, weaker than the proximal; and further, if at all severe, it is rarely limited to one limb. Simulated palsies, on the contrary, are frequently limited to a single segment of a limb, as the fingers; or they may appear as total inability to move one limb, with the rest of the movements of the body intact. And the disability, when due to organic causes, is constant; we cannot by any means obtain more voluntary movement, or the exertion of greater power, than the patient professes himself able to make. In cases of functional or simulated weakness, however, it is usually easy to see that the patient has more power than he believes he has or professes to have when tested directly: a limb that is apparently powerless may be held up for a moment or two if the support is suddenly withdrawn from it, or if the fingers only are weak the patient may be seen to use them, in dressing or undressing himself, in a manner in which a man with an equal degree of organic paresis could not do. Another valuable point of distinction is that, when asked to exert as much power as he can, the organic patient does his best; he contracts the muscles at once as firmly as he can, maintains the contraction until they tire, and the amount of power exerted varies with the mechanical conditions under which the muscles work: the fingers, for instance, can be flexed more strongly and through a greater range when the wrist is extended than when it is flexed. The hysteric and the simulator, on the contrary, give the impression that they are not doing their best; they exert power slowly and intermittently, and frequently it may be increased considerably by persuasion or by suggestion. And the strength of the movements usually bears no relation to the mechanical conditions under which the muscles contract: the grasp may be as strong when the wrist is fully flexed as when it is extended. But the most characteristic feature of functional or simulated weakness is that the patient usually contracts a series of muscles that should not be concerned in the intended movement, or which may actually impede it; when asked to straighten his elbow, for example, all the muscles of the limb may stand out in firm contraction, including the biceps, which normally and in every form of organic paralysis relaxes when the triceps contracts in order to extend the joint. This contraction of the antagonists of the muscles that should shorten is very common, it is in

fact the means by which the simulator frequently feigns weakness, but it occurs in hysteria too. Further, lesions of the central nervous system produce paresis, which, though at first flaccid, becomes spastic within a few weeks—that is, the tone of the weakened muscles increases, contractures or shortenings of certain muscles are liable to develop, and the hypertonicity impedes passive movements. Functional or simulated weakness, on the contrary, is never spastic, and the voluntary muscular contractions by which the patient may resist passive movement are easily distinguished from spasticity.

The state of the *reflexes* is one of the most important guides we have in deciding whether a disability is due to organic nervous lesion or not. Disease of the peripheral nerves leads to disappearance of the corresponding tendon-jerks, which are never absent when the nervous system is intact. Too much weight is, however, frequently laid on exaggeration of the knee-jerks and other tendon reflexes, for they may be as brisk in hysterical and other states as they ever are in organic disease, and the distinction of organic clonus from pseudo-clonus is so difficult that often it is reliable only in the hands of an expert. The superficial reflexes—that is, the plantar and abdominal—are more valuable, since an extensor plantar response invariably means organic disease, and a unilateral or local absence of the abdominal reflex has the same significance (*see REFLEXES*). The *sensory* disturbances which are simulated or are of hysterical origin differ both in distribution and in character from those produced by organic disease. The injury of a nerve causes loss of sensation in the area to which its fibres are distributed, a spinal lesion an alteration or loss of sensation below the corresponding root area, and one in the brain-stem or forebrain an anæsthesia on the whole or on part of one side of the body. The simulator, on the contrary, complains of loss of feeling in the whole of one limb, or in one or more of its segments, as below the elbow or knee or on some other area of skin—that is, the anæsthesia has a distribution that cannot be explained by the anatomical arrangement of the sensory fibres. The *character* of the loss is even more significant, as there is frequently complete cutaneous insensibility though the sense of position and other forms of deep sensation remain intact; such a dissociation is never due to organic nervous lesions, though these may produce dissociated anæsthesia of

other types. (*See SPINAL CORD, LOCAL LESIONS OF; and NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF.*)

For the distinction between true and simulated *disturbances of the special senses* the reader must be referred to special treatises; but he may be reminded that a unilateral blindness, a common complaint of the malingerer, is, when due to organic disease, associated with a disturbance of the reaction of the pupil to the light, and generally with the changes in the media and fundus which can be detected by the ophthalmoscope; and that lesions behind the chiasma produce homonymous defects in the visual fields which the simulator cannot copy.

As the malingerer complains so frequently of pain, a short paragraph is necessary on the *differentiation of true from simulated pain*. This is often a matter of extreme difficulty. Frequently the malingerer's story raises suspicion: his pain is in many cases constant and invariable, while that due to physical causes is rarely so; and it may be described as very intense or intolerable, though his appearance and general health suggest that he has not suffered severely. On examination we frequently find that, while the patient's attention is directed to the seat of pain, all the muscles in the neighbourhood, as around a joint, are contracted so firmly that if an organic lesion were present it would excite pain; in disease certain muscles only are, as a rule, tonically contracted in order to limit movement. Then we usually find that even the slightest touch in the region of simulated pain aggravates it: the man who asserts he has pain in his back following a spinal injury cries out whether we merely touch or raise a fold of skin gently, or squeeze the muscles, or press on the bones; or if it be headache, raising the scalp gently by pulling on the hair excites a show of agony. Finally, we can usually discover that the pain is not aggravated by those causes that would increase it if it were due to the injury to which the patient attributes it: stretching the nerve by Lasègue's or Neri's method does not usually make the pain in simulated sciatica worse; and with a little tact the malingerer with a painful spine can be usually made to flex, extend, or rotate his vertebral column without discomfort.

2. Independence of simulated symptoms of any disease that may be present.—The malingerer is often not a sound man, or the accident to which he attributes his symptoms may have

produced organic changes, though these are not responsible for them. Factitious symptoms are frequently superimposed in real disease, or the malingering may take the form of culpable exaggeration. The separation of the real from the assumed is often a matter of great difficulty which cannot be discussed in detail here. It should be always a general principle to investigate the organic changes as thoroughly as possible, then analyse the functional disturbances that they might produce, evaluate their total effect, and see in how far this is compatible with the symptoms which the patient presents.

3. Distinction of malingering from hysteria and the psychoneuroses.—This is the hardest task that confronts the medical officer, as there is no single sign which enables him to distinguish the simulator from the hysteric, or from the patient with a traumatic neurosis; it is mainly on his experience that the expert must rely, but prolonged observation is often necessary before a decision can be arrived at. The following points, however, should be borne in mind. The symptoms of hysteria are relatively constant and are frequently found unchanged during a series of successive examinations, while those that the malingeringer presents generally vary considerably, especially if he is under constant observation. In hysteria the complaints are usually more or less systematized and definite, but those of the malingeringer are usually vague and indefinite, though he may of course simulate a single symptom, as blindness, or loss of power in a limb. Further, the simulated symptoms are not amenable to mental or moral influences, and particularly to suggestion, as are the manifestations of hysteria. And in malingering we never find those vaso-motor and secretory disturbances, and wasting of the muscles of a disused limb, which are often associated with the gross physical symptoms of hysteria. The attitude and demeanour of the patient is often significant too; as a rule the hysteric is candid and self-confident, as he is convinced of the reality of his troubles. He usually co-operates with and aids the medical man in his investigations, while the malingeringer is often sullen and passively resistive; he overacts the part and takes every opportunity of insisting that his disability is so obvious and severe that investigation is superfluous. It has been rightly pointed out that the hysteric revels in examination, while the malingeringer loathes it and frequently does what he can to avoid or post-

pone it. Finally, the discovery of the so-called hysterical stigmata, as contraction of the visual fields, cutaneous anæsthesia of the segmental type, and the detection of other functional disturbances of which the patient was unaware, and to which he apparently attaches no importance when they are demonstrated, may be important signs in separating the honest claimant from the malingeringer.

4. Positive evidence of malingering.—We can rarely obtain legal proof, or demonstrate objectively, that a man is malingering, though we may be convinced of it. Careful observation sometimes reveals a definite discrepancy between his complaints and his behaviour. A man who professes inability to use his arm may be seen to move it when his attention is distracted, or even to employ it in dressing or undressing himself, or in regaining his balance if it is threatened; he may bend his back though he complains of a rigid spine, or react to a weak auditory or visual impression though he says he is deaf or blind. But it is frequently only by taking the malingeringer unawares, or by placing him under observation of which he knows nothing, that we can obtain evidence of simulation that will be accepted by the law.

GORDON HOLMES.

MALPRAXIS.—A term which by common usage is attached to the act of failing to exercise reasonable skill, knowledge, and care in the treatment of patients. It is not a statutory offence, and there are no special laws dealing with it. It is, however, an offence against the common law of the land, and there are numerous judicial decisions dealing with the subject in its various aspects. A civil action for the recovery of damages is the remedy of the aggrieved person, or, if the facts warrant it, a criminal prosecution may result, or conceivably both procedures might be instituted. It is, I feel certain, not sufficiently appreciated by members of the medical profession at large that, however correct may be their conduct, and however skilful, painstaking, and careful their treatment, they are always at the mercy of any person who may feel aggrieved. No permission has to be obtained before bringing an action for malpraxis, and thus the worry and expense of a defence at law may descend upon any practitioner, however meticulously careful he may have been. In such an action there is usually a slight substratum of fact, and upon this flimsy foundation a superstructure of allegations and mis-

representations is easily reared. In all probability the action will be unsuccessful, but that is a very negative form of comfort. At the best, the practitioner stands where he did, having sustained all the worry, extra work, and loss of time and money, and knowing that if the plaintiff is a man of straw he will be left to pay his own costs. Should the plaintiff, on the other hand, be successful, the result might well be ruinous, damages and costs easily mounting into large figures.

I have made this aspect prominent because I desire to urge that every medical man ought to be a member of one or other of the mutual defence associations. There are such things as blackmailing actions, and there are solicitors who will act for plaintiffs when the grounds of action are of the most flimsy character, in the hope that a monetary consideration for the stay of proceedings may be forthcoming. The knowledge that a powerful association is ready to take up the defence is a very healthy deterrent to the bringing of frivolous actions.

The Legislature has recognized the peculiarly dangerous position of medical men with regard to the bringing of actions after the signing of certificates and other matters in connexion with the administration of the Lunacy Acts. To found an action, facts pointing to want of good faith and absence of reasonable care must be adduced. When proceedings are commenced they may, "upon summary application to the High Court or a judge thereof, be stayed." But even in this matter the right of litigation ought not to be absolutely withheld from any person feeling aggrieved. Actually very few such cases have come into court.

With regard to the question of the amount of skill and care which a medical practitioner must bring to bear upon a case, it is obvious that there can be no hard-and-fast rule. The fact that the medical practitioner is registered within the terms of the Medical Acts is a presumption that he is possessed of the skill and knowledge which the law requires. The position is well set forth in the following judgment: "He who undertakes the public practice of any profession undertakes that he has the ordinary skill and knowledge necessary to perform his duty towards those resorting to him in that capacity."

The legal wrong which the patient sustains consists in having received incompetent or negligent treatment, and the onus is upon the plaintiff to prove this as a fact. It is obvious

that there can be no arbitrary standard of skill, and, given the presumptive competence of the practitioner—i.e. the fact that he possesses a registered qualification—the amount of skill he is expected to bestow is that ordinarily possessed by the average practitioner. In general terms, the degree of skill expected from a medical or surgical practitioner was thus laid down by Lord Chief Justice Earle: "A medical man is certainly not answerable merely because some other practitioner might possibly have shown greater skill and knowledge." He must "have a skill and knowledge which is undefinable, but which must be a competent degree in the opinion of the jury."

It is thus plain that the uncertainties of the law have ample scope for disconcerting vagaries in this class of action. Each case has to be decided upon its own merits. The amount of skill which it would be reasonable to expect from a surgeon or physician practising in a large town, and having opportunities to keep himself *au fait* with current and recent methods of treatment, cannot reasonably be expected from one practising in a country village far remote from the centres of experiment and research.

As to specialists, it has been laid down that "the duty of a specialist is referable to a higher test than that of an ordinary practitioner. Special profession invites higher duty, and the standard to be attained is that of the specialist and not that of the general practitioner, and this includes proper instructions to the nurses and to the patient for their conduct in the intervals of the doctor's attendance."

These remarks as to after-treatment and the giving of instructions apply equally to the specialist and to the ordinary practitioner. A dislocation may be skillfully reduced, but if specific instructions are not given it may recur, or the joint may become permanently stiff because movements are not initiated. Such untoward events might be held to indicate a lack of reasonable attention and care.

A point on which a good deal of misconception exists is the liability of a medical man to a patient whom he treats gratuitously. It must be borne in mind that the legal relations of doctor and patient are not altered thereby. "Whether the service is remunerated or gratuitous is immaterial."

The adoption of a method of treatment which is new, or not universally recognized by the profession, may result in an action for damages if the issue be unfavourable or attended by

untoward results. Where there is a divergence from the system of the majority, the jury have to determine whether the practitioner is a scientific inquirer or a mere ignorant pretender. If a notable departure from recognized methods is contemplated, and especially if failure to achieve the desired result may be evidenced by unfavourable objective signs, the fullest confidence between doctor and patient should be established, all possible contingencies explained, and a full and free consent obtained. The practitioner's knowledge of his patient and of human nature in general must guide him in deciding whether to obtain such consent in writing. This would very rarely be necessary.

When an operation has to be performed on a young person or on someone who is in a position of dependency, the consent of a responsible person should always be obtained. It may save a great deal of subsequent explanation, and it rarely means more than a little initial extra trouble. The practice of inoculation encountered opposition before the War; but its beneficial effects were then so apparent that little antagonism remains. It is when an alleged bad result is taken up by an "anti-" society anxious to increase its membership (and incidentally its funds), or by some local politicians desirous of securing notoriety, that trouble usually arises.

Actions have been brought when, because of an idiosyncrasy, the exhibition of a drug has had unusual and harmful results. The intolerance of some persons towards belladonna may be cited as an illustration. In my own experience an action was brought because the application of a belladonna plaster was followed by the appearance of an intensely painful, persistent, and widely distributed rash. In a similar case it was stated in judgment that "it would be a dreadful thing if a man were to be called in question criminally whenever he happened to miscarry in his practice."

In the early days of X-ray medication, actions for the recovery of damages were brought in cases in which burns or baldness followed application of the rays. They belong to the period when no means of estimating the dosage of the rays was available. Should a similar untoward result occur nowadays it would be necessary to prove that reasonable care and skill (in the light of present knowledge) were exercised in administering the treatment, so that the abnormality must be due to an idiosyncrasy of the patient.

In order that criminal proceedings may be taken against a medical man, extreme carelessness or gross ignorance must be shown to have occurred, and they would not be commenced until after careful consideration by the Crown. In one case a practitioner was charged with having caused the death of a woman while delivering her with forceps, being at the time under the influence of alcohol. It was shown that his mental condition was due to chloral hydrate and not to alcohol, and he was sentenced to three months' imprisonment.

Criminal charges of malpractice are uncommon, but civil actions in which damages are claimed for negligence are more frequent. In my experience, the phrase "reasonable skill and care" is interpreted by the judicial bench in a manner quite tolerant, and even lenient, towards the medical practitioner. But though a judge may take a favourable view of professional skill and attention, the attitude of a jury is by no means certain. The defendant's case has therefore to be fought with full determination.

Where a specific act of malpractice is alleged, the defendant may not produce evidence that he is a skilful person. The point at issue is not what he is capable of doing, but what he has actually done. Evidence of skill is admissible, however, when there is a dispute as to whether the specific act is malpractice or not. If the specific act is admittedly malpractice, then the question whether the practitioner has skill which he did not use is irrelevant. *Prima facie*, it is negligence to leave an instrument or a swab in a wound after an operation, but even here the whole circumstances must be reviewed.

Want of skill must be in the treatment itself, and not in the treatment with reference to the particular constitution of the patient, unless the treatment presupposes that knowledge. Thus it is not malpractice to apply a belladonna plaster, even if untoward consequences ensue, unless the practitioner is aware that the patient has an idiosyncrasy against belladonna; in that event the treatment becomes negligence.

Improper treatment may be a ground of defence to an action for fees for professional attendance. A practitioner cannot recover fees for the cure of a wound which he himself has caused through his misconduct.

Finally, a medical practitioner does not undertake that his treatment is infallible;

hence, if he sues for his fees, it is no defence to allege that the treatment was ineffective and mistaken, unless it was negligently or ignorantly so, nor that it failed of effect, nor that the opinion he formed after examination was mistaken.

F. S. TOOGOOD.

MALTA FEVER (see UNDULANT FEVER).

MANIA.—Mania is a form of insanity characterized by excitement, mutability, divertibility, and frequently by exaltation. It is almost the antithesis of melancholia, in which there are dullness and sadness. It has been noted, however, that many attacks of mania are preceded or followed by attacks of melancholia, or, where the condition is recurrent, the expected attack of mania is replaced by one of melancholia. For these reasons many authors now include mania and melancholia as subdivisions of one disease, manic-depressive insanity (q.v.). Whatever may be the scientific value of such an arrangement, from the point of view of treatment it is well to keep the two subjects, mania and melancholia, distinct.

Etiology.—States of excitement account for 10–15 per cent. of the admissions into mental hospitals. Defective heredity is present in the greater number of those in which a satisfactory history is forthcoming. Young adult life furnishes the most cases, but the disease occurs at all ages. Mental shock, worry, financial and other losses, have been stated to be the etiological factors in certain cases, but in many instances no definite exciting cause can be found.

Pathology.—Neither the naked eye nor the microscope reveals any definite changes in the brain of patients dying from mania. The general opinion is that the condition is due to a toxæmia of some kind.

Symptomatology.—The varieties are (a) simple mania, (b) acute mania, (c) chronic mania, (d) mania transitoria, (e) acute delirious mania.

In (a) **simple mania** the patient is talkative and restless. His whole nature is changed; for example, the quiet man becomes boisterous, careless of his company, lacks reticence, and his talk is suggestive. He is, as a rule, in a joyous condition, except when through some trivial cause the happiness gives place to anger. Sleep is diminished. There are no hallucinations or delusions. This may be the whole illness, from which the patient may recover, or may pass into acute mania.

(b) **Acute mania.**—Here the mental symptoms just described appear in an exaggerated form. The patient is excited; he shouts, sings, laughs, and occasionally cries, only to laugh again shortly afterwards. There is complete loss of self-control, with perhaps violence, destructiveness, and immodesty. He is continually talking, and the content of his conversation exhibits that form of incoherence known as “the flight of ideas.” Word follows word in a constant stream with no apparent link, but, if the whole is taken down in shorthand, associations can be found connecting the ideas that find verbal expression. If a question is put sharply to the patient a fairly rational and correct answer can often be obtained, but it is only a momentary gleam of reason, and he is off again into his ramblings. The general behaviour is similar to the speech; there is incessant activity which is constantly changing.

An important diagnostic point is that the patient, both in conversation and behaviour, is easily diverted into other lines of thought or action. He is sleepless and refuses food, is neglectful of his person, and is often very dirty in his habits.

(c) **Chronic mania** may arise out of the two preceding varieties, if neither death nor recovery takes place. The excitement grows less, but traces of it remain. It is a condition of craziness, frequently with delusions. It does not run a regular course. Outbursts of extreme excitement may occur at irregular intervals.

(d) **Mania transitoria** is a rare condition, in which a short, sharp attack of acute mania runs its course in a few days.

(e) **Acute delirious mania** is usually described under the heading of mania, but in my opinion a separate compartment should be found for it as a febrile disorder with delirium. It resembles acute mania, but differs from it in certain respects. In acute delirious mania the cause is often definite, the onset frequently sudden, the temperature is raised, there is more persistent refusal of food, greater restlessness and insomnia, greater incoherence of speech and disturbance of consciousness, and pronounced hallucinations (Savage). The treatment of this condition resolves itself into maintaining the strength of the patient by tube feeding. Stimulants also should be given. It is frequently fatal.

The physical signs of mania include loss of weight, general ill-health, furred tongue, com-

MANIA

stipation, anæmia, a quick and feeble pulse, low blood-pressure, and coarse tremor of face and hands. The excessive movements occur mainly at the proximal joints. There is frequently a leucocytosis.

Course and prognosis.—Simple mania frequently ends in recovery. If not, it gradually merges into acute mania. Acute mania, after lasting three months or so, also frequently ends in recovery. In rare cases the attack is so acute that exhaustion supervenes and death occurs, sometimes with alarming suddenness. If neither death nor recovery takes place, the condition becomes chronic. Even here recovery may take place after many years. Signals of ill omen are the development of unchanging delusions and symptoms indicative of commencing dementia. These are loss of memory and the formation of faulty habits.

Differential diagnosis.—*General paralysis* often commences in an attack which is very like acute mania. Physical signs of the former disease must be looked for in every case of mental disorder. The excitement which occurs in some cases of *dementia præcox* is similar to acute mania. In the former disease some rigidity, or the presence of stereotypies or mannerisms with emotional deterioration, will assist in the diagnosis. It must be remembered that epileptics occasionally have attacks of furor, a condition not unlike acute mania.

Treatment.—It is rarely possible to nurse the patient at home, so arrangements should be made to send him to a mental hospital as soon as possible. At first, till the excitement subsides, he may have to occupy a padded room, but as soon as possible he should be taken out and placed in bed in the open air. The prolonged hot bath is a valuable sedative. The diet should consist of light and easily digestible food and plenty of it. Should food be refused no hesitation must be felt in resorting to tube-feeding. The danger to life lies in exhaustion, and most patients will recover if sufficient food is assimilated. Drugs should be used as little as possible. When the excitement is intense and there is danger that the strength of the patient may succumb, sedatives will be required as the lesser of two evils. Hyoscine hydrobromate, given hypodermically in doses of $\frac{1}{16}$ to $\frac{1}{8}$ gr., is a powerful and useful drug. In less severe cases a dose of chloral 20 gr., with 30 gr. of mixed bromides, is frequently of service. For sleeplessness,

MANIC-DEPRESSIVE INSANITY

paraldehyde, sulphonal, trional, or veronal should be used. As convalescence becomes established, tonics will be required. Aperients should be given to combat the constipation present. For further details, see *INSANITY TREATMENT OF*.

R. H. STEEN.

MANIA, ACUTE DELIRIOUS (see p. 259 and under *CONFUSIONAL INSANITY*).

MANIC-DEPRESSIVE INSANITY.—In the earlier writers the simple classification of insanity into the three divisions of mania, melancholia, and dementia was the one usually adopted. Mania included those cases in which the predominant features were excitement, noise, loquacity, and unruly behaviour. In melancholia the patient was sad, quiet, and rarely spoke. In dementia there was enfeeblement of thought, feeling, and will.

With careful clinical study it soon became evident that though mania and melancholia appear to be as diverse as possible, yet they possess many bonds of union. For example, certain attacks of mania are ushered in by an attack of melancholia, or an attack of mania ends in melancholia. Furthermore, it was noticed that mania and melancholia are both liable to recur. These recurrences may be at such regular intervals of time as to be definitely periodic, though more commonly they occur at irregular intervals.

It was further noted that the recurrences were not always of the same type. For example, an initial attack of mania might be recovered from, then a relapse occur with another attack of mania from which recovery ensued; and when the next breakdown came, instead of mania there was an attack of melancholia. There are other cases in which the attacks follow one another in sequence or cycle; for example, mania is followed by melancholia, after which there is a remission of symptoms, and then again mania, followed by melancholia and remission, and so on throughout the lifetime of the patient.

This condition is called *folie circulaire* (q.v.), or circular insanity. Recurrent mania is the name given to the condition when an initial attack of mania is followed, after remission, by further similar attacks; in recurrent melancholia a series of attacks of melancholia separated by remissions occurs; and in alternating insanity an attack of mania is followed by a remission, this is succeeded by a phase of

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melancholia, then a second attack of mania, and so on.

Stupor, which is a state of enfeeblement or paralysis of the mental functions from which recovery is possible, sometimes follows mania or melancholia, or replaces either, or is itself recurrent. (See STUPOR.)

There is thus the large group Manic-depressive Insanity, with the subdivisions (1) Mania, (2) Melancholia, (3) Circular Insanity (Folie Circulaire), (4) Manic-depressive Stupor.

It is to be noted that there are two varieties of melancholia. One is the depressive stage of manic-depressive insanity; the other is the depression of later life called involutionary melancholia. The condition known as stupor is also met with in other diseases—for example, in dementia præcox, hysteria, epilepsy, and general paralysis of the insane.

The above is a short summary of the teaching of Kraepelin and his school. This has been largely adopted in America and on the Continent of Europe, and by the younger generation in England.

It should be pointed out that there are objections to this conception, which may be given in a few words. In the life-history of an individual an attack of mania or melancholia has been known to occur followed by recovery and no recurrence. The view, therefore, that the attack is only the first of a series is apt to lead to an unnecessarily gloomy prognosis. Furthermore, when the illness is in progress it has to be combated on definite lines if mania, on other lines if melancholia. The conception does not therefore assist in any way in the treatment of the patient. An account of each of the subdivisions of manic-depressive insanity is given under the appropriate headings, to which the reader is referred.

R. H. STEEN.

MARASMUS, INFANTILE (Atrophy, Athrepsia, Decomposition).—The term marasmus is used in diverse ways by differing authors. As employed in this country it usually signifies wasting (its true definition), or failure to gain weight, though certain authors restrict it to a state in which, as the result of chronic dyspepsia, the body-cells are unable to metabolize food. It will be considered here in its wider sense.

Etiology.—Deficient power to digest and assimilate may be congenital in origin, and in this regard the health and environment of the mother and the length of her family are factors

of importance. *Premature and weakly infants* and those with congenital defects clearly stand a poorer chance of thriving than do others. *Syphilis* is a prominent cause, for it leads to premature birth as well as to the birth of infants ill prepared to utilize food; very pronounced degrees of malnutrition are found in congenital syphilitics. *Tuberculosis*, either in the family or in the patient, must be mentioned as an occasional but much less common cause. By far the most important causes, because at once the most common and the most preventable, are *dietetic*. Continued underfeeding or overfeeding, premature weaning, too frequent or irregular feeding, and food unsuitable for the particular child, or indeed for any child, must be included under this head. Marasmus of this order is usually preceded by continued *digestive disturbance* for which one food after another has been tried, without precision or cogency, until the powers of digestion and assimilation are crippled beyond repair, or only recuperated by slow and laborious steps. In chronic underfeeding, on the other hand, marasmus often precedes the alimentary symptoms. Among the causal dyspeptic disorders must be noted an intolerance of special food elements which is inadequately met (see *Chronic Dyspepsia of Infants*, under *DIARRHEAL DISORDERS OF INFANTS*). *Summer diarrhoea* may eventuate in marasmus, which may cause a fatal issue long after the more acute condition has been overcome. When these common causes have been mentioned, it remains to be said that any serious organic disease of long standing will cause in infants, as in adults, chronic wasting and malnutrition. Among these, because particular to infants, must be noted congenital hypertrophic stenosis of the pylorus (see *PYLORUS*, *CONGENITAL HYPERTROPHIC STENOSIS OF*).

Symptoms.—In appearance the marasmic child is a miniature presentment of an old man. He is thin, shrivelled, and below normal weight, and mental and physical development are retarded. The eyes and fontanelles are sunken, the hair sparse and often broken or worn away in the occipital region. The skin is dry and harsh, grey or muddy in hue, and, being loosely applied to underlying structures, hangs in folds about the face, thighs, buttocks, and elsewhere. The face and forehead are wrinkled, the cheeks hollow, though the sucking pads stand out in considerable relief. The bones are prominent, while at points of pressure the skin becomes reddened and sometimes

MARASMUS, INFANTILE

inflamed, and bedsores may follow. Excoriation is often seen in the napkin area, due to irritating discharges. Diarrhoea or bulky undigested stools are passed, perhaps containing mucus. Vomiting is generally present and may be severe. The tongue is dry and red, and stomatitis is common. The abdomen is usually protuberant and tympanitic, but may be carinate. The circulation is poor, the extremities easily becoming blue and cold, while the tips of the fingers are often shiny. The temperature is low, often subnormal. The blood shows a considerable degree of secondary anæmia. The child is fretful, restless, and irritable, but later becomes apathetic. The lymph-glands are often prominent, and the valves of superficial veins can easily be located by the presence of minute swellings along their course. Peritæal hæmorrhages, œdema of the feet and hands, and a falling temperature herald a fatal issue. Among complications, stomatitis, boils, otitis media, bronchitis, broncho-pneumonia, pulmonary collapse, intractable vomiting, and terminal gastroenteritis are noteworthy.

Treatment.—The primary aim should be to determine the cause of the wasting. In the case of weakly infants with small powers of digestion, maternal nursing should be continued whenever possible. Failing this, a wet-nurse is the best substitute. When neither is available, milk should be given in small quantities, predigested and freely diluted; if the infant cannot suck, it may be given by a spoon or dropper. Should it fail to be digested, it may be necessary to give albumen water, whey, or a malted food temporarily. Warmth is essential, and an incubator may be used. The infant should be wrapped in cotton-wool and gently lubricated with olive oil or cocoa butter daily. Alcohol is very valuable, in doses of 5 min. every three or four hours. Saline may be necessary if collapse threatens. The food must be kept low should the infant survive, the amount of fat being especially restricted. In the case of older infants, some error will usually be found in the dietary. The food ordered will depend largely upon the condition of the stools. In amount it should always be well below the normal for the age and size, and a careful watch must be kept on the stools and the weight, for the combined observation of these affords the best indication whether the diet is sufficient or whether any particular element is ill borne. As the child makes up for lost ground the diet may gradually be approximated to the normal. In cases in

MASSAGE

which the marasmus originates from particular digestive disorders the treatment must be modified accordingly, and is essentially that of the causal condition. Syphilis, hypertrophic stenosis of the pylorus, or other determining causes demand their appropriate treatment.

FREDERICK LANGMEAD.

MASSAGE.—In its modern form, this treatment may be said to have been originated about a hundred years ago by the Swede P. H. Ling, in connexion with his plan of using exercises in the treatment of disease. Most of the massage manipulations now used were included among his so-called “passive-movements,” but their present-day French designations (*effleurage*, stroking; *pétrissage*, kneading; *friction*, frictions; and *tapotement*, tapping, clapping, or hacking), as well as the term massage itself, were only introduced some fifty years later by Prof. Mezger, of Amsterdam. A variation of the tapotement, *vibrations*, has of late come into extensive use owing to the invention of various mechanical appliances for producing the desired rapid percussion, or lateral or rotatory movements, according to the different types of vibrators.

Space does not allow of a detailed consideration of the different physiological effects of the various manipulations, nor of the way in which they should be carried out. This is best learnt by demonstration and practice, but as general rules it may be stated that the operator should let as large a surface as possible of his hand be in contact with the part worked upon, in order to gain the maximum effect with the minimum of exertion to himself and pain to the patient; and that in all the manipulations, except *effleurage*, the patient's skin should move as much as possible with the operator's hand so that the effect may reach the deeper structures and the skin be saved from irritation.

In practice, the various manipulations merge naturally into one another, and in experienced hands there should never be much consideration whether this or that manipulation should be used, but rather what effect is aimed at and what anatomical structures are worked upon.

The term massage is often taken to include passive and even active movements up to a certain point not very well defined, as these are generally used in close and intimate connexion with it. Theoretically, the distinction is that any performance which involves an

action of the patient's muscles and joints constitutes a movement, whether carried out by the operator while the patient remains passive, or performed by a voluntary action on the part of the patient.

Trauma in various forms, such as contusion, strain, sprain, dislocation, and fracture, constitutes one very important field of usefulness for the masseur. One object of the massage in these conditions is to remove the extravasated blood in the tissues and the effusion which is likely to take place in joints. In slighter cases the treatment may be started at once; but if more extensive tearing of blood-vessels has occurred, it is safer to wait for some twenty-four hours in order to give the bleeding time to stop. When an injured joint is in a state of acute synovitis with the effusion still increasing, it is safer to postpone the massage for a couple of days. In any case, it should be commenced merely as a light *effleurage*, confined to the neighbourhood of the injury, and more especially to the region proximal to it, so as to reduce the congestion of the injured part by emptying the veins and lymph-vessels. This will lessen the existing tension and consequent pressure upon the fine nerve-endings, and thus pain will be relieved, making it possible gradually to extend the manipulations nearer and nearer to the site of injury and to include the more deep-reaching *pétrissage*. Later on, and in cases that do not come under treatment until some time after the injury, more energetic measures in the form of firm frictions are necessary to break up and disperse the collections of extravasated blood and lymph then organizing, and to promote the absorption of the persisting synovial effusion in joints and tendon-sheaths. These manipulations are very important, for they tend to prevent the formation of adhesions and to restore the normal function of the injured part. It is at this juncture that the passive and active movements above referred to come in as an essential part of the treatment, not only to assist in preventing adhesions, but also to keep the capsule and ligaments of joints from contracting and to maintain the nutrition of the muscles concerned.

This improved nutrition naturally has a pronounced beneficial influence also on the repair of bones in cases of **fracture**. The treatment of fractures by early massage and movements has been brought very much to the front in recent years, especially through the work and writings of the late Prof. Lucas-Championnière

of Paris, who laid particular stress upon the sedative influence on the sensory nerves of the very light but regular and continued *effleurage* with which he always initiated the first examination and the subsequent applications of the treatment. This helps to relieve pain and consequently to allay the muscular spasm which is such a marked and undesirable feature in cases of fracture, since it gives rise to much additional pain and is liable to cause displacement of fragments. Throughout this method of treating fractures the guiding principle is to pay the greatest attention to the soft parts involved in the injury, so as to restore the functional utility of the limb affected, the repair of the bone being at the same time thereby promoted.

Another great class of surgical disorders in which massage is usefully employed is that of **deformities**, and among them **spinal curvatures**. In these spinal cases massage would be used in combination with exercises, and, in comparison with them, it usually plays a minor part in the treatment. The principal object of the massage proper is to strengthen the weak musculature of the back, to remove pain, whether due simply to a feeling of fatigue or to pressure on the intercostal nerves, and to relieve contraction of the overacting muscles. The comparative values of the massage and the exercises in a particular case depend upon the severity of these features. Thus, in the beginning of a course of treatment one has generally to rely more upon the massage; whilst as the patient's strength increases the exercises gradually become more important.

With regard to the particular manipulations to be used in these circumstances, *effleurage* and *vibrations* are most effective in relieving pain and muscular contraction; *effleurage* and *pétrissage*, by their influence on the circulation, help to improve the nutrition and consequently the strength of the weakened muscles (i.e. principally on the convexities of the curvatures); and *tapotement*, acting as a typical mechanical stimulus, tends to increase the power of contractility of the muscles.

By being subjected to these various procedures the muscles are placed in the most favourable condition for responding to the impulse of the will. Voluntary contraction should therefore follow upon the massage, the increased arterial flow produced in the muscle by its own action following up the advantage that has been gained by the previous emptying

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of its veins and lymph-vessels and the removal of accumulated fatigue products.

This is the principle upon which massage, combined with movements, acts upon weak and overworked muscles generally; and it should furnish guidance in the treatment of deformities, not only of the spinal column, but also of the extremities, where the action of the opposing sets of muscles is often even more obvious.

Just as massage is employed with great advantage in the treatment of accidental injuries, so also it may frequently be used after various **surgical operations** in order either to follow up and complete the object aimed at by an operation or to counteract any undesirable effect of the surgical interference.

How local conditions of this nature may be dealt with has already been sufficiently indicated in connexion with injuries and deformities, but some additional consideration ought to be given to the very important general effect upon the system which is achieved by massage. By improving the circulation, stimulating the metabolism, restoring the muscular tone, increasing the activity of the skin, and in other ways, this treatment powerfully assists recovery after an operation or any other debilitating condition.

This general effect of massage is perhaps still more frequently taken advantage of in the treatment of **medical diseases**, even in cases where it cannot be expected to have any real influence on the disease itself. There are, however, many medical disorders in which massage forms a valuable direct aid to cure, or at least to an alleviation of the symptoms.

It is obvious that many affections of the **circulatory organs** must derive benefit from the efficient help that can be rendered to the circulation by its means. Naturally, the peripheral parts of the circulatory system are those that most need, and that are most accessible to, this external aid to the circulation. This applies not only to the extremities but also to the portal system, for massage of the abdomen is particularly useful in this connexion. The effect is not only local but is seen throughout the whole circulatory system, in a diminution of venous congestion and an increase of arterial blood-pressure. The relief to the work of the heart thus brought about results in slower and more complete contractions, and is of considerable practical value in the treatment of various cardiac diseases. In addition, we have at our disposal other manipulations

which influence even more directly the action of the heart. Such are tapping of the back and chest, which lowers the pulse-rate (presumably by stimulating the vagus), and stroking, fine vibrations and rhythmical tapping over the cardiac region, which act upon the nervous ganglia within the heart itself. In all these cases massage should be employed, as usual, in conjunction with passive and active movements of the extremities and trunk in order further to facilitate the circulation and to strengthen the heart-muscle by carefully graduated increased exertions.

Breathing exercises constitute our chief mechano-therapeutic means of influencing disorders of the **respiratory organs**, but massage is not without its uses in this connexion. *Chronic catarrh* of the pharynx and larynx, for instance, is often greatly benefited by repeated administrations of effleurage and vibrations over the throat, *chronic bronchitis* and *emphysema* by tapotement of the chest. The latter affection is also favourably influenced by pressure and vibrations applied at the time of expiration in order to make this process more complete, partly by the mechanical compression of the thoracic cavity and partly by stimulating the contractility of the elastic tissue in the lungs.

The **abdominal organs** are particularly accessible to the manipulations of massage, as has been pointed out in another connexion. It was then mentioned how effectively the whole circulatory system could be influenced by the aid given to the portal circulation, and it stands to reason that there must be a very distinct local effect at the same time. By the lessened congestion and the fuller supply of arterial blood throughout the mucous membrane of the alimentary canal, the functional activity of its glands is increased; the same applies to the liver and pancreas. Absorption of the products of digestion is facilitated by the increased circulation. The muscular tissue in the intestinal wall profits by the improved nutrition, and at the same time its fibres are induced to contract by the mechanical stimulation, the result being an increased peristalsis and a gradual improvement of any existing state of dilatation of the stomach and intestines. Similarly, the muscles of the abdominal wall gain in strength, adipose tissue is reduced, and the size of the abdomen lessened by the massage. In addition, it exerts an important direct mechanical influence on the contents of the intestines, breaking them up and forcing

them onwards—an action which is particularly effective in the colon. The principal manipulations used to achieve these various objects consist of deep pétrissage of the whole of the intestines and incidentally of the abdominal wall, tapotement for the liver, and also over the stomach to induce muscular contraction of its walls, friction to break up and dislodge accumulated masses in the colon (most likely to occur at the hepatic and splenic flexures), and, finally, deep effleurage along the course of the colon.

The complaints most obviously benefited by this treatment are *constipation* and cases of *gastric dilatation* and *visceroptosis*, but many of the *chronic catarrhal conditions* can also be improved by a suitable variation of the manipulations.

As is well known and recognized, numerous affections of the **nervous system** benefit by massage. To take one example, it very materially assists recovery in cases of *hemiplegia* by maintaining the muscles in the best possible state of nutrition, preventing the occurrence of contractures, and stimulating the conductivity of the nerves by means of so-called nerve frictions, i.e. quick, sharp movements across the nerve, carried out with the point and nail of one finger or thumb in places where the various nerves are accessible between the muscles, and preferably where they are passing over bone. This mechanical stimulation often produces visible contractions of the corresponding muscles. The combined effect of the massage of muscles and of the stimulation of the nerves helps to facilitate the natural muscular response to the impulse of the will. In affections of the lower motor neurones the difficulty of maintaining the nutrition of the parts involved gives added importance to the massage, stimulating manipulations like tapotement being then included in the treatment. On the other hand, when spasticity of muscles is a marked feature, tapotement, nerve frictions and vibrations should be avoided, and more quietening measures employed, such as stroking and slow deep kneading. Thus varied and adapted to the different circumstances, massage has a very important and useful mission to fulfil in the treatment of a great variety of nerve diseases.

RICHARD TIMBERG.

MASTICATORY SPASM (see TRISMUS).

MASTITIS (see BREAST, AFFECTIONS OF).

MASTOIDITIS.—A purulent infection of the mastoid process.

Etiology and pathology.—Mastoiditis is practically always secondary to suppuration of the middle ear. That mastoiditis does not occur in every case of acute suppuration of the middle ear is apparently due to the fact that some mastoid processes contain few air-spaces, and so do not readily lend themselves to abscess-formation. There is, however, one mastoid air-cell—the mastoid antrum—which is never absent, even in non-cellular mastoids. It is involved in probably all cases of middle-ear infection. But whatever pus may be formed in it generally drains away, with the pus in the middle ear proper, through the opening in the membrane after perforation. When this happens there is no danger of further extension, and the antrum and the middle ear recover together. Occasionally, however, the exit of pus from this small cell is obstructed, and it is compelled to seek an outlet elsewhere.

If the mastoid process contain no pneumatic cells, the pus formed in the antrum may break forth from its prison by passing outwards between the bony wall of the external auditory meatus and its cutaneous lining, until it reaches the exterior of the skull just above the auricle in the region of the temporal muscle. Here it collects and forms a temporal abscess. In other cases, particularly those of children, the pus may liquefy the bone of the mastoid superficial to it, and so pass out to collect under the periosteum of the mastoid process behind the ear, the typical mastoid abscess. If, on the other hand, the mastoid process be filled with cells, these become infected, and osteitis with pus production and the formation of granulation tissue results. The granulations absorb the partitions between the cells until the whole of the mastoid process becomes converted into a unilocular cavity filled with granulation tissue and pus.

Next follows an extension of the disease beyond the limits of the mastoid process. A common extension is to the groove of the lateral sinus. This vessel becomes protected by a covering of granulations, and the infection fails to reach its interior in the vast majority of cases. Occasionally, however, the defences are imperfect and thrombophlebitis is set up (see THROMBOTIC PHLEBO-ARTERITIS). Another frequent extension is to the superficial aspect of the mastoid process, where it forms the characteristic mastoid abscess behind the ear. This region is reached by means of a fistulous

opening on the outer surface of the mastoid process. Sometimes the fistula forms on the same surface, but nearer to the tip of the mastoid. In that case the pus reaches the sterno-mastoid muscle and, being contained within its sheath, sets up a limited cellulitis in this region, forming Bezold's abscess.

Symptoms.—After the opening of the membrane in a case of acute suppuration of the middle ear and the appearance of a free discharge of pus by the meatus, the pain continues, the temperature remains above 99° F., pressure with the finger over the bone behind the ear demonstrates great tenderness, and some oedema is also usual at this site. The temperature ranges between 99° and 100° F. If it rise above 100° F., lateral-sinus thrombosis or labyrinthitis should be suspected.

When the pus breaks out from the mastoid, an abscess forms in the soft tissues corresponding in position with the site of the fistula. Thus, with a temporal abscess, swelling and oedema with deep fluctuation appears in the scalp above the ear, and the auricle is projected downwards and outwards by the swelling. When it forms in the mastoid region the local signs of abscess appear behind the ear, and the auricle projects directly outwards with little or no downward tendency. Lastly, in Bezold's abscess there are swelling, redness, and tenderness in the region of the upper third of the sterno-mastoid muscle, with pain and stiffness on attempted movement of the head.

Diagnosis.—When an abscess has formed, diagnosis is easy, but it is very important to be able to recognize mastoiditis before the abscess makes its appearance. Attention to the signs given above will prevent any oversight. The most important of them are pyrexia and tenderness. In the course of middle-ear suppuration a temperature of 99° F. or more, after the ear has begun to discharge freely, should lead to operation. (For the distinction between mastoid abscess and furuncle of the external auditory meatus, see p. 439.)

Treatment.—If an abscess has already formed when the patient is first seen, it should at once be opened by a skin incision. Then arrangements should be made for a mastoid operation. If the disease be diagnosed before abscess in the soft parts has formed, a mastoid operation should be performed as soon as possible. In uncomplicated mastoiditis the prognosis after operation is good, and it is often possible to restore the hearing.

DAN M'KENZIE.

MASTURBATION.—The frequency of self-abuse is impossible to compute, but it would appear to be no very unusual feature of school life in both sexes. Like other bad habits, it is usually learnt from others, and therefore the extent to which it is practised varies greatly in different schools or other institutions. Instruction by other children or the evilly disposed is not necessary, however, as is instanced by the little-realized frequency of the habit in small children—girls more often than boys—at ages varying from a few months to a few years of age. This should be kept in mind when a mother complains that her small girl baby will not sit still, flushes and sweats profusely, and afterwards turns pale. In some cases local irritation, as by threadworms or vulvitis, appears to originate the practice. It is relatively infrequent in adult life. In its most repeated and serious form it is a manifestation of mental disorder, and thus is probably explained the popular belief that masturbation leads to insanity.

The consequences of the habit are seen unalloyed in the case of infants, who become pale and flabby, petulant, ill-tempered, and perverse. Appetite is diminished or lost, and physical and mental attainments are retarded. In older children and adults a sense of moral degeneration may be added and bring with it self-consciousness and secretiveness. Though there can be little doubt that repeated self-abuse causes exhaustion with its attendant train of symptoms, such as lack of concentration, deterioration of work, lassitude, and the like, yet the boggy of self-destruction has been held too threateningly before the delinquents in the past and has done much to deepen their depression, weaken their effort to overcome the habit, and imbue them with a painful and paralysing sense of their own degradation which may have far-reaching results.

The doctor should enter the patient's life as a friend and not as an inquisitor or monitor. He must make it clear that the patient is not a moral leper to professional eyes. Once the right atmosphere is created, talks and encouragement will often lead to reassertion of the patient's control and cessation of the habit. As accessory measures the patient may be advised to take regular cold baths morning and evening, to keep both body and mind employed by work and exercise, and to rise immediately on waking in the morning. The diet should not be stimulating, nor the bed too comfortable. Numerous drugs have been advocated, notably

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the bromides, brometone, gelsemium, and black willow (*Salix nigra*); in themselves they are of little use, but they may be employed to enhance the value of such rational psychotherapy as is indicated above.

In the case of boy infants circumcision, and in that of girl infants splinting or blistering, may be successful.

FREDERICK LANGMEAD.

MAXILLARY ANTRUM, DISEASES OF (see SINUSES, ACCESSORY AIR, DISEASES OF).

MEASLES (*syn.* Morbilli; Rubeola). — An acute contagious disease characterized by catarrh of the upper respiratory passages and a blotchy eruption.

Etiology.—Though the specific organism has not been discovered, there is little doubt that measles is due to a microbe. Positive results have often been obtained by inoculation of healthy subjects with blood from cases of measles. The unknown infective agent is contained in the discharges from the mouth and nose and is disseminated by coughing, sneezing, talking, etc. The disease is conveyed mainly by direct transmission and rarely by fomites or by third persons. Schools play an important part in its spread. That the infectivity of the disease is highest in the prodromal and eruptive stages, and then rapidly ceases, is proved not only by clinical evidence but also by experiments on animals.

Measles is the commonest of all infectious diseases and is widely distributed throughout the world. It is more prevalent in towns than in the country, especially in densely populated areas. It is most frequent from November to January, and again in May and June. Though no age is exempt, it is essentially a disease of childhood. W. Butler, from examination of nearly 14,000 children in elementary schools, found that by the age of 15 nearly 97.3 per cent. had been attacked. The disease is rare during the first six months of life, but congenital cases are not unknown (see p. 269). This relative immunity ceases during the second six months, and the highest morbidity is in the second to the seventh year of life. When populations hitherto exempt are attacked, as occurred in the Faroe Islands in 1846, and in Fiji in 1875, adults are as susceptible as children.

Bacteriology.—The virus of measles has been shown by Anderson and Goldberger to possess the following characteristics: (1) It

can pass through a Berkefeld filter. (2) It can resist drying for 25½ hours and freezing for 25 hours. (3) It loses its infectivity after 15 minutes at 55° C. (4) It may possibly retain some infectivity after 24 hours at 15° C.

Various micro-organisms act as secondary invaders. Thursfield has shown that the most important of them is the streptococcus pyogenes, which gives rise to a septic condition of the mouth, fauces, and naso-pharynx, and is the most frequent cause of death towards the middle or the end of the second week. Infection with the pneumococcus is more rapidly fatal, causing death at the end of the first or beginning of the second week. *B. influenza* plays a part in the production or prolongation of broncho-pneumonia, which, after septicæmia, is the most frequent cause of death. Staphylococcal infection is less frequent. In cancrum oris and noma putridi the fusiform bacillus and the spirillum of Vincent predominate.

Pathology.—The principal lesions are found in the skin and mucous membranes of the upper respiratory tract. The morbid process in both cases is essentially the same, and consists in vascular dilatation and diapedesis of leucocytes round the vessels and glands. The toxins of the disease cause coagulation necrosis of the superficial cells, and desquamation results. The inflammation of the mucous membrane of the upper respiratory tract usually ends in resolution, but in some cases ulceration of the laryngeal mucosa occurs, with involvement of the subjacent cartilage. The intestine may be the seat of changes similar to those in the respiratory tract. In fatal cases areas of focal necrosis have been found in the liver.

In view of the close relationship supposed to exist between measles and tuberculosis, it should be noted that Thursfield found no evidence of pulmonary or glandular tuberculosis in careful autopsies on 34 cases of measles.

The blood.—During the incubation period there is a marked increase in the neutrophils, but leucopenia is the rule both in the prodromal and in the eruptive stages. On the occurrence of a complication such as broncho-pneumonia or stomatitis, leucocytosis develops.

Symptomatology.—The incubation period is usually 10–14 days, but may be as short as 6 or as long as 25 days. As a rule, it is usually symptomless, but a considerable lowering of the body-weight, starting on the fourth or fifth

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day and lasting several days, has been described (Meunier's sign).

The **prodromal period** comprises that which intervenes between the end of the incubation period and the appearance of the eruption. It is usually three full days, but may be curtailed when there has been a recent acute infection, especially scarlet fever.

The principal symptoms of this period are rise of temperature, catarrhal signs, rashes, and Koplik's spots.

The characteristic *temperature* of the prodromal period is intermittent. From normal it rises to a height ranging from 99° to 102° F. at night, and falls again to normal in the morning. There is usually little constitutional disturbance, but young children may be fretful or drowsy.

The *catarrhal signs*, which rarely appear before the rise of temperature, consist of a frequent cough—which may be croupy—laryngism, sneezing, and nasal discharge. The fauces are injected, and there may be some complaint of sore throat. These signs soon become more pronounced as one mucous membrane after another becomes affected, and reach their height at the time of the eruption.

Before the appearance of the specific eruption, however, various *prodromal rashes* may occur, assuming the form of isolated macules, blotchy or scarlatiniform erythema, or urticaria. Two or more of these varieties often coexist. They are to be found on any part of the face, trunk, or limbs, and do not, like the prodromal rashes of smallpox, especially affect the abdomino-femoral region or the axillæ. They cause no local irritation. Their duration varies from a few hours in the case of urticaria to one or two days in the case of scarlatiniform erythema or isolated macules. Prodromal rashes are usually associated with other symptoms of the pre-eruptive period, but may constitute an isolated phenomenon of a precatarrhal stage of measles.

Koplik's spots.—These spots, described by Koplik in 1896, are bluish-white specks surrounded by a red areola which usually appear first on the buccal mucous membrane opposite the first molar teeth. They are not found on the palate or on the gums. Only a few may be present, or the whole buccal mucous membrane may be covered with them. They precede the specific eruption by two or three days, and begin to fade soon after it appears. They do not occur in any other condition, and this fact, taken in conjunction with their almost in-

variable appearance in the pre-eruptive stage, invests them with the greatest possible diagnostic value.

Comby has given the name of erythematopultaceous stomatitis to the gingivitis which precedes and accompanies the eruption. The gums are swollen and red, and often covered with a thin whitish coating. This sign, unlike Koplik's spots, is not peculiar to measles, being found in scarlet fever, influenza, and other acute diseases. It is not present, however, in rubella or other morbilliform eruptions.

Eruptive period.—Usually on the fourth day from the onset the specific eruption appears, in the form of small, discrete, amorphous red macules behind the ear, and on the chin and upper lip. During the next twenty-four hours it spreads over the face, neck, scalp, and arms. Finally the back, abdomen, and limbs are involved. (Plate 20.) The individual lesions become larger and distinctly papular, and frequently crescentic in shape. As a rule, the eruption becomes semi-confluent on the face, which assumes a characteristic puffy appearance, while on the trunk and limbs the lesions are generally more discrete. The eruption is usually more copious and develops earlier round pre-existing skin lesions than elsewhere. It begins to fade from above downwards, and by the time it has reached its full development on the lower limbs the lesions on the face have usually faded. Its complete duration is from three to five days, but may be as short as a few hours or as long as six days. As it fades it leaves a brown or purple staining, which may persist for a fortnight and serve to establish a retrospective diagnosis of measles.

Desquamation is usually flaky or branny in character, and may be absent. On the other hand, it may be almost as profuse as in scarlet fever. It rarely lasts for more than a fortnight. The palms and soles usually escape.

The eruption is always accompanied by an aggravation of the catarrhal symptoms and an increase of pyrexia. The constitutional disturbance varies. The tongue is usually furred, there is loss of appetite, and diarrhoea is frequent. The temperature frequently rises to 103° or 104° F., and continues with only slight remissions at this high level throughout the eruptive stage. The fall to normal is usually rapid as the eruption fades.

Varieties.—Measles assumes anomalous forms less frequently than any other acute exanthem. Toxic and hemorrhagic cases on the one hand, and abortive attacks on the



PLATE 20.—MATURE MEASLES ERUPTION.

(Ricketts and Byles.)



other, are much less common in measles than in scarlet fever or smallpox.

Toxic measles.—The principal features of toxic measles are early and severe prostration, hyperpyrexia, and a rapidly fatal course. The rash is not prominent, and may be absent.

Hæmorrhagic measles.—A form characterized by purpura and hæmorrhages from the mucous membranes, and running a rapidly fatal course, is extremely uncommon. Probably some of the cases so described were really examples of hæmorrhagic smallpox. A petechial eruption, especially at the elbows, axillæ, and other flexures, is not uncommon, even in mild cases, and has no sinister significance.

Morbili sine morbillis, or rubæola sine exanthemate, is an abortive attack in which all the other symptoms are present except the specific eruption, viz. catarrh, Koplik's spots, pyrexia, and even prodromal rashes as in a case reported by the writer. This variety is most likely to be met with in mild epidemics, especially if the patient has acquired a partial immunity by a previous attack.

Rubæola sine catarrho was the name given by Willan to a disease in which there was a complete absence of catarrh, ophthalmia, and fever, although the course and appearance of the eruption were those of ordinary measles. Although the catarrhal symptoms in measles may be more severe in some cases than in others, their entire absence is so exceptional that Willan's cases were probably examples of rubella.

Congenital measles.—Ballantyne has collected 20 cases of fetal measles. In each case the infection in the mother and fœtus was simultaneous. According to Rocaz, the attack is always very mild, and the rash does not reach the lower part of the body.

Measles in infants.—In weakly and cachectic infants the rash may be very incomplete in its distribution, and around areas of irritation, such as are caused by intertrigo or eczema, may be more prominent than elsewhere. The prognosis is worse than in older patients owing to the frequency of respiratory complications.

Relapses and second attacks are rare. One attack usually conveys immunity for the rest of life.

Complications. Respiratory system.—*Laryngitis* occurs in the great majority of cases at some stage of the disease. It is usually catarrhal, but may be ulcerative or membranous. In the prodromal and eruptive periods it is usually due to pneumococci or streptococci.

Laryngitis of later onset is more likely to be diphtheritic. In catarrhal laryngitis the symptoms as a rule are mild, and consist only of a hoarse cough, a husky voice or complete aphonia; in ulcerative and membranous laryngitis dyspnoea is a prominent symptom, often requiring intubation or tracheotomy. Some degree of tracheitis and bronchitis is constant.

Broncho-pneumonia is the most important complication. It is most likely to occur below the age of 3 years, and accounts for 50–90 per cent. of the deaths from measles. Its frequency varies in different epidemics, but is always greater in institutions than in private practice. It is usually due to the streptococcus pyogenes, less frequently to the pneumococcus and the influenza bacillus. The complication develops on the second or third day of the eruption, but may occur after it has faded. Its onset is revealed by a rise of temperature and sometimes by a sudden disappearance of the eruption. The respirations become rapid and laboured, the pulse is quickened, and there is a frequent short cough. Dullness may not always be detected on percussion, but fine moist râles can usually be heard. In favourable cases the temperature falls to normal within a week or ten days, but the disease may be protracted for six weeks or more as one area of the lung after another becomes involved. The mortality is high. Of 670 measles cases admitted to the Grove Hospital of the M.A.B., 103 developed broncho-pneumonia, of whom 53, or 51·4 per cent., died; the oldest fatal case was 6 years, and all but three were under 5 years. The sequels of broncho-pneumonia, in order of importance, are pulmonary tuberculosis, gangrene, fibrosis, and emphysema.

Lobar pneumonia is much less frequent than broncho-pneumonia, and is more likely to be found in older children and adults. *Pleurisy*, serous or purulent, is usually associated with broncho-pneumonia, but it may occur independently.

Digestive system.—*Catarrhal stomatitis*, especially affecting the gums, is constant, and rapidly subsides as the rash fades. *Ulcerative stomatitis* is more important. The commissures and inner surfaces of the lips are chiefly involved; the ulcers are round or oval, and may be covered with a greyish membrane resembling diphtheria, but yielding only cocci on cultivation. *Gangrenous stomatitis*, or *cancrum oris*, though said to be more frequent

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following measles than after any other acute exanthem, is nowadays decidedly uncommon. It was more frequent in past times when overcrowding, defective nursing, and general neglect of sanitary precautions were rife. If the patient survives, considerable deformity results from cicatrization, but the complication is usually fatal.

Tonsillitis.—During the prodromal and eruptive stages older children and adults often complain of a *sore throat*, and some swelling of the tonsils and patchy deposit may be seen. Pseudo-diphtheria or ulcerative sore throat is an occasional sequel of measles. The tonsils, uvula, and palate become covered with membrane resembling that of diphtheria, but ulceration rapidly takes place and often spreads to the larynx. No diphtheria bacilli are found on cultivation, but only streptococci or staphylococci. Death takes place from septicæmia, or from hæmorrhage owing to erosion of a large vessel in the pharynx or larynx.

Gastritis may occur and cause troublesome vomiting, but is less frequent than *enteritis*. Next to broncho-pneumonia *diarrhœa* is the most important complication. It chiefly affects marasmic infants, but may prove serious even to adults. It is most liable to occur during the hot months of the year. The pathological condition is a catarrhal enteritis or enterocolitis. The process may become chronic.

Skin.—Herpes facialis may occur during the early stage. Miliaria, sudamina, and petechiæ frequently accompany the specific eruption. Other accidental rashes, such as urticaria, and scarlatiniform or blotchy erythema, may be post-eruptive as well as prodromal. Impetigo, boils, and abscesses are not infrequent in convalescence. Gangrene in other areas of the skin than those involved by cancrum oris and noma pudendi is sometimes seen. Subcutaneous emphysema may be associated with broncho-pneumonia or be found apart from any obvious pulmonary lesion, when it has been attributed to rupture of the bronchi or larynx as the result of violent coughing. Disseminated tuberculosis of the skin, lupus vulgaris, and erythema nodosum occasionally follow measles. The cuti-reaction to tuberculin is invariably negative during the eruptive period (energy), but the sensitiveness to tuberculin rapidly returns as the rash fades. Ordinary vaccination performed on the day preceding the eruption and the first two days of the eruption shows a similar energy (Netter).

Genito-urinary system.—Aronson and Som-

merfeld have shown that the urine in measles contains a thermostable dialysable toxin which proves virulent to guinea-pigs or rabbits on intravenous injection. They found that the toxicity of the urine bore no relation to the severity of the disease, and that the urine from other infectious diseases, such as scarlet fever, typhoid fever, tuberculosis, diphtheria, and whooping-cough, contained no toxin. The diazo-reaction is positive in about 80 per cent. of cases during the eruptive period, and often remains so for a few days after the rash has faded. Albuminuria may occur, especially during the febrile period. Hæmaturia and other signs of nephritis are rare. Bacilluria due to *B. coli* may occur and clear up rapidly, but John Thomson has drawn attention to an extremely chronic type almost entirely confined to late childhood, which is especially apt to follow measles. Vulvo-vaginitis is not uncommon, and may occur even in mild attacks. Noma pudendi, a gangrenous process affecting the external genitals, is as rare as the analogous process, cancrum oris.

Cardio-vascular system.—Though less common than in scarlet fever, myocarditis may occur, especially in severe cases, and be the cause of sudden death. Endocarditis is rare, and is more frequently diagnosed during life than verified post mortem. Galop has reported a case of infective endocarditis in which paresis of the upper limb and gangrene of the lower limb on the right side were due to embolism. Pericarditis may occur, chiefly in association with pleuro-pneumonia. A few cases of venous or arterial thrombosis in the limbs have been reported. All were fatal.

Lymphatic glands.—Some degree of cervical adenitis is constant, though not so marked as in rubella. Suppuration rarely takes place. Both the cervical and the bronchial glands may undergo chronic enlargement and form a nidus for tubercle bacilli.

Nervous system.—A large number of nervous complications have been attributed to measles, often without sufficient justification. In some the original diagnosis of measles was doubtful, in others the nervous sequel occurred too long after measles to be causally connected with it, and in others again the coexistence of diseases more liable to attack the nervous system, such as diphtheria or syphilis, had been overlooked. The brain is more frequently attacked than the spinal cord or the peripheral nerves. Convulsions may occur at the onset, but are not common. Epilepsy may date from an attack

of measles. Cerebral palsies may appear in the form of hemiplegia or diplegia or affect individual nerves. Encephalitis is the characteristic lesion, being more frequently found after measles than after any other acute exanthem. Cerebral or cerebellar abscess may arise in connexion with otitis or, much less frequently, rhinitis. Meningitis is rare, and either is due to otitis media or is one of the manifestations of tuberculosis following measles. Optic neuritis may occur in connexion with or apart from meningitis. A few cases of ocular palsy have been recorded. Other nervous complications sometimes observed are myelitis, disseminated sclerosis, peripheral neuritis, chorea, and tetany.

Special senses.—*Otitis media* is one of the most frequent complications: it is most liable to develop in young persons and after severe attacks. It may be catarrhal or purulent. Two varieties of the former are described. The first, which occurs in the eruptive period, is less a complication than a localization of the rash in the mucous membrane of the middle ear: it is mild in character and subsides without perforating the tympanic membrane. The second variety occurs at a later period, and is due to spread of infection from the naso-pharynx by the Eustachian tube. The otitis then usually becomes suppurative and causes perforation of the tympanic membrane. The micro-organisms found in the discharge are usually streptococci and pneumococci. The lesions as a rule are less severe than in scarlet fever, but mastoid abscess and necrosis of the ossicles may occur. Deaf-mutism may follow spread of the infection to the internal ear.

Catarrhal conjunctivitis is a constant accompaniment of measles. It usually clears up as the eruption fades, but in rare cases becomes chronic. Blepharitis and sties are not uncommon, especially in ill-nourished children. Keratitis is a more serious complication. Corneal ulceration is liable to occur, and to lead to permanent opacities or to be followed by perforation of the cornea and panophthalmitis.

Bones and joints.—Bone complications are rare, and usually secondary to cancrum oris, in which necrosis of the upper and lower jaw may ensue. Acute infective periostitis is an occasional sequel of measles. The joints are very rarely involved. Adults are chiefly affected, the joint trouble appearing either as arthralgia or as an acute simple or suppurative

arthritis. Tuberculous bone- and joint-disease may first develop after measles.

Association with other infectious diseases.—Measles may precede, accompany, or follow any of the other infectious diseases, but is most frequently associated with whooping-cough. The coincidence of diphtheria and measles is very serious. Not only is the larynx liable to be involved, but the disease often assumes a malignant form, and is little affected by large doses of antitoxin. Broncho-pneumonia almost invariably supervenes.

Although measles is usually credited with predisposing to tuberculosis or lighting up a pre-existent infection, tuberculosis was noted as a sequel in only 45 (0.42 per cent.) of a M.A.B. series of cases. Therefore, either the current view as to the relation between measles and tuberculosis is incorrect, or tuberculosis does not occur till a late stage of convalescence.

Diagnosis.—Errors of diagnosis may be made both before and after the appearance of the eruption. In the prodromal stage measles may be mistaken for acute rhinitis, bronchitis, or influenza on account of the catarrhal signs, or for diphtheria on account of the laryngeal cough, or for scarlet fever on account of the prodromal scarlatiniform rash. These errors may be avoided by inspection of the buccal mucous membrane and bacteriological examination of the throat. The presence of Koplik's spots and the absence of diphtheria bacilli will indicate the true nature of the disease.

In the eruptive stage a confusion is most likely to be made between measles and rubella, as is shown by the fact that among 656 cases admitted with the erroneous diagnosis of measles to the M.A.B. hospitals, 319 were suffering from rubella. The eruption in the two diseases may be closely alike, but in rubella Koplik's spots are absent, and catarrhal signs, even if present, are ill marked.

Measles may closely simulate the papular eruption of smallpox, especially on the face, but the distribution and development of the eruption in the two cases, the temperature—which is high in the eruptive period of measles and comparatively low in the papular stage of smallpox—and the absence of Koplik's spots in smallpox are sufficiently distinctive.

Morbilliform rashes due to drugs, such as copaiba, chloral, or quinine are not usually accompanied by fever or catarrh, and Koplik's spots are invariably absent.

Prognosis.—The prognosis depends chiefly

on the age and the previous health of the patient, ranging in different epidemics from 3 to 50 per cent. The mortality is highest during the first two years of life and then rapidly falls. It is always higher in hospitals than in private practice, owing to the large number of young and ill-nourished children admitted.

Broncho-pneumonia and enteritis are the most serious complications, and the association of diphtheria with measles is particularly unfavourable. The tendency of measles to predispose to tuberculosis should be borne in mind.

Treatment.—The patient should be kept in bed in a room whose temperature should not be below 60° F., and be given a fluid diet during the febrile period. Bright light should be excluded to protect the eyes, which should be bathed frequently with warm boric lotion or with a 10-per-cent. solution of argyrol. Special attention should be given to the mouth, nose, and throat, for the disinfection of these parts is the best prophylaxis of septicæmia, broncho-pneumonia, and otitis. The laryngitis may be treated by hot fomentations to the neck, but if dyspnoea is urgent, tracheotomy or intubation will be required. In addition to the ordinary treatment for broncho-pneumonia, vaccine-therapy should be instituted according to the results of the blood-cultures. For diarrhoea, Dover's powder or bismuth salicylate is useful. The local application of salvarsan affords the best results in cancerum oris.

Within a week after the temperature has become normal and the catarrhal signs have ceased the patient should be allowed up, and in another week to go out of doors if the weather be favourable. Care should be taken to shield the convalescent from the infections of whooping-cough, diphtheria, and tuberculosis, to which he is specially liable.

J. D. ROLLESTON.

MEASLES, GERMAN (*see* RUBELLA).

MEAT POISONING (*see* Food Poisoning, under POISONS AND POISONING).

MEDIASTINUM, AFFECTIONS OF.—The mediastinal affections considered here are mediastinitis in its various forms, and tumours of the mediastinum.

SIMPLE ACUTE MEDIASTITIS

This may occur in association with severe cases of acute pericarditis and acute pleurisy, and lead to adhesions between the chest-

wall and the pericardium, and between the pericardium and the pleura. It is hardly recognizable clinically, apart from the other diseases present, but occasionally "mediastinal crepitation" can be heard on deep inspiration; sometimes its time is that of cardiac rhythm.

SUPPURATIVE MEDIASTITIS

This is a rare condition, due usually to extension from tuberculous mediastinal glands or from disease of adjacent bones, or else to burrowing of pus from the neck, as in cases of angina Ludovici or after tracheotomy. It is occasionally met with in general septic conditions, especially with malignant cases of the acute specific fevers, and with septic pneumonia or pleurisy. The pus either infiltrates the mediastinal tissues or forms a localized abscess in the anterior or, more rarely, in the posterior mediastinum. Eventually it either points on the surface of the chest or ruptures into a neighbouring viscus—the air-passages, lung, pleura, œsophagus, or pericardium. The duration varies from a few days to several months.

Symptomatology.—The most frequent symptom is increasing pain, with tenderness, behind the sternum or between the shoulders; this may be paroxysmal or pulsating in character. A localized abscess may cause pressure symptoms, such as cough, dyspnoea, dysphagia, and signs of venous obstruction. The general symptoms are those of suppuration and sepsis. If an abscess points, it gives rise to superficial redness and œdema, followed by a fluctuating swelling at the top of the sternum or in an intercostal space. Pulsation may be communicated by the aorta to the swelling, which may thus closely simulate an aneurysm. If the pus ruptures into the air-passages, it may either be coughed up or cause death from suffocation; if into the œsophagus, it may be vomited up.

Diagnosis may be very difficult, unless there is some obvious cause for the disease, as symptoms may be almost latent.

Prognosis.—The disease is almost necessarily fatal, unless the pus can be let out in good time, and is therefore especially serious when the posterior mediastinum is involved or when there is diffuse suppuration.

Treatment.—The main object is to evacuate the pus as soon as possible. Earlier treatment is entirely symptomatic; the strength should be maintained, and pain relieved by

MEDIASTINUM, AFFECTIONS OF

hot applications externally and, if necessary, by opiates internally.

GANGRENOUS MEDIASTITIS

This form is met with very occasionally, and is nearly always due to ulceration and perforation of the œsophagus by a foreign body or by malignant disease. The symptoms are indefinite unless a communication is established with the air-passages, in which case they are very similar to those of gangrene of the lung. The condition is inevitably fatal.

CHRONIC MEDIASTITIS

Chronic mediastinitis results from an acute mediastinitis should this subside, and is usually associated with adherent pericardium. It may be chronic from the first, and is then generally secondary to tuberculous disease of the bronchial glands or lungs. (*See PERICARDITIS; POLYTORRHOMENITIS.*)

TUMOURS OF THE MEDIASTINUM

Mediastinal growths of almost every kind are met with, but they are generally malignant—sarcomata (especially lympho-sarcomata) in young people, and carcinomata in later life. They mostly start in the mediastinal glands, and may be either primary or secondary, sarcomata being usually primary. Growths commencing in the thyroid gland sometimes extend into the mediastinum. In lymphadenoma any enlargement of the thoracic glands is usually merely a part of a general lymphatic overgrowth. Tuberculous disease of these glands, though very common, rarely gives rise to the signs or symptoms of a definite mediastinal tumour, except occasionally from pressure on the trachea or bronchi.

Symptomatology.—Symptoms are mainly due to pressure and are naturally very similar to those produced by aneurysm. Pain is often present, but varies much in severity, character, and distribution. Dyspnoea and orthopnoea are common, either from direct pressure on the trachea or main bronchi, in which case they are attended by stridor and whistling or wheezing sounds, or from pressure on the vagus or recurrent laryngeal nerves, giving rise to laryngeal spasm or to paralysis of the abductor muscles of the vocal cords. With tracheal obstruction there is absence of the respiratory excursions of the larynx. Hoarseness may be present independently of dyspnoea. Cough is sometimes troublesome and, if stridulous, croupy, or clang-

ing in character, is very suggestive of pressure on the trachea. Expectoration is usually scanty or absent unless there are secondary changes in the lungs. Hæmoptysis may result from invasion of the lungs or air-passages by the growth, or from pressure on the pulmonary veins. Pressure on the superior vena cava and its main affluents is common in tumours of the anterior mediastinum, and causes lividity, swelling, and œdema of the head, face, neck, or arms. Dysphagia, which may culminate in complete obstruction, is met with occasionally from pressure on the œsophagus.

General symptoms are not often pronounced, but wasting and anæmia are common in the later stages, and fever may be present in cases of lymphadenoma, or as a result of secondary changes in the lungs.

Physical signs vary in different cases within very wide limits. No one sign is pathognomonic, and almost any abnormal sign may be present. A small growth may compress the trachea and cause death with few or no signs; a large one near the surface may give quite unmistakable signs. In addition to signs due directly to the growth, others of the most varied character may result from involvement of the air-passages and lungs, for in the latter there may be overdistension, collapse, fibrosis, œdema, low forms of pneumonia, bronchiectasis, and gangrene. Another frequent complication is a pleuritic effusion, which may be bloodstained.

On inspection and palpation, special points to note are—dilatation of the superficial veins, enlargement of external lymphatic glands, especially of those above the clavicles and in the axillæ, and any alteration, local or general, in the shape of the chest or in the respiratory movements. Sometimes there is local bulging, but occasionally one side of the chest is contracted. The heart may be displaced in any direction. Vocal fremitus may be either increased, diminished, or lost.

Percussion over a superficial growth gives a dull note, with greatly increased resistance. The dullness often extends across the middle line, differing in this respect from that of an uncomplicated pleural effusion. Over one or both lungs the resonance may be modified in various ways, according to the secondary changes set up by the growth. Similarly, the breath-sounds vary greatly, being sometimes weak or even absent, sometimes bronchial or blowing. The

vocal resonance also varies correspondingly. Stridulous sounds are produced by narrowing of bronchi, and moist sounds by destructive changes in the lungs.

Diagnosis may either be obvious or extremely difficult. When signs and symptoms of an intrathoracic tumour are present it is necessary to distinguish between an *aneurysm* and a solid growth. If the patient is a man of middle age engaged in a laborious occupation the probabilities are in favour of aneurysm, especially if he has suffered from syphilis; in women and in young or old men there is far greater likelihood of a solid growth. The pressure symptoms of aneurysm are apt to be more variable, owing to alterations in its size; and whilst an aneurysm presses more on the nerves and gives rise to severe pain, a solid growth is more likely to compress the large veins, producing œdema, etc. An X-ray examination may settle the diagnosis, but it must be remembered that a growth may have pulsation communicated to it by the aorta, and so resemble an aneurysm both in the screen examination and on ordinary inspection and palpation of the chest. Enlargement of the external lymphatic glands is, of course, a most valuable diagnostic sign. When, as is not uncommon, the signs of lung disease are prominent, the presence in addition of distinct pressure symptoms may indicate the existence also of a mediastinal growth. In cases in which there is a pleural effusion the real diagnosis may first be revealed by the persistence of well-marked signs after withdrawal of all the fluid. Cancer-cells may also be found in the fluid. It may for a time be almost impossible to distinguish between *syphilitic stricture of a bronchus* and a growth at the root of the lung too small to give rise to characteristic physical signs. The nature of a mediastinal growth must often be altogether uncertain, but in many instances the indications point very definitely to a secondary carcinoma or to a lymphadenomatous tumour.

Prognosis is nearly always most unfavourable; the duration of life is generally shorter than in cases of aneurysm.

Treatment, as a rule, can only be palliative, pressure symptoms being relieved by measures similar to those employed in cases of aneurysm. If there is the smallest possibility that the disease may be syphilitic, suitable treatment should, of course, be pushed vigorously.

J. WALTER CARR.

MEDICAL EVIDENCE AND REPORTS ON MEDICO-LEGAL CASES.—Medical men may be required to appear as witnesses in both criminal and civil cases. For purposes of description medical evidence may conveniently be divided into:

1. *Documentary evidence*: (a) Medical Certificates, (b) Written Reports, (c) Written Opinions, (d) Dying Declarations (q.v.).
2. *Oral or parole evidence*.
3. *Experimental evidence*, e.g. when, in the case of gunshot wounds, experiments have been conducted to show the possibility of the wound having been produced by a particular projectile and from a particular distance and direction.

Both documentary and experimental evidence must eventually be deposed to in the witness box.

Documentary evidence.—*The medical certificate* is the simplest form of documentary evidence, as when a registered medical man grants a certificate stating the inability of a person (witness or juror) to attend court by reason of illness. Such a certificate should bear the address at which it is written and be dated; it should also bear the name and address of the person for whom it is given and state the nature of the illness preventing attendance, and should be signed with the name and degrees of the writer. In Scotland such certificates must, in addition, bear the words "on soul and conscience."

Written reports are given chiefly at the instance of the Public Prosecutor, and their contents will vary with the nature of the case. Such reports will be a statement of medical facts observed by the writer, and deductions drawn therefrom.

The following general rules should be observed in the drawing up of reports:—

- (1) The date and place of the report should be stated, also the hour, day, month, and year of the examination.
- (2) The age, sex, and occupation of the person reported on should be included when applicable.
- (3) The medical facts should be concisely stated in plain simple language which can be understood by a non-medical person. Where the use of technical terms is necessary their meaning should be given in brackets. Indefinite terms such as "about" or "nearly" should be avoided: thus in the case of wounds

MEDICAL EVIDENCE AND REPORTS ON MEDICO-LEGAL CASES

the actual measurement should be taken. Again, such an expression as "considerably decomposed" should be replaced by a precise description of the conditions found.

(4) The report should conclude with a reasoned opinion based on the facts observed.

(5) The report should be signed with the name and medical qualifications of the examiner, and in Scotland must, like medical certificates, be attested "on soul and conscience."

In the case of post-mortem examinations the preamble of the report should state on whose authority it was made, and the place of the examination. The names, designations, and addresses of any persons who may have identified the body in the presence of the examiner should also be included. The results of the external examination should first be given, and then the results of the examination of all the cavities and all the organs should be stated *seriatim*. Even when a possible cause of death has been discovered, the complete examination is still necessary.

Written opinions on a case may be given at the request of counsel—generally in civil cases. These may be used simply for the guidance of counsel, or the medical man may subsequently be required to appear in court as a witness (expert).

Oral or parole evidence.—On entering the witness-box, the witness is sworn to tell the truth, either by "kissing the book" or in the Scots fashion by uplifting the right hand and repeating after the judge, "I swear by Almighty God as I shall answer to God at the great Day of Judgment that I will tell the truth, the whole truth, and nothing but the truth." By Section 5 of the Oaths Act, 1888, a witness has the option in *any* English court of taking the oath after the Scots fashion. On hygienic grounds the advisability of doing so need not be debated.

Medical men usually occupy the position of skilled or expert witnesses, as they are generally called upon to give evidence depending on technical knowledge. But, like other people, they may occasionally be called as ordinary witnesses to testify to facts which have been observed by them, e.g. as witnesses of an assault. In such cases, questions involving medical or surgical knowledge should not be asked. If the witness is required to answer such questions, then he becomes a skilled witness, and should be remunerated accordingly. In giving evidence he should bear in mind certain principles:

(1) He should not be a partisan. His skilled evidence is required to assist in the administration of justice.

(2) His answers should be clear, concise, and free from ambiguity, and technical terms should be avoided. If he does not know, he should state so distinctly, and should refuse to be bullied by cross-examination into modifying his statement. He should remember that considerable licence is allowed to counsel, and should not be intimidated or irritated by the questions put, or the manner of putting them. He will sometimes be asked to answer a question by a simple "Yes" or "No," and if the question does not admit of such an answer he should appeal to the judge to allow him to make an explanatory statement.

(3) He is not allowed to quote from medical or surgical works, but counsel may read a passage from some recognized textbook, and ask him if he agrees or disagrees with the statements there made. In such cases it is well to ask to see the book, as it is not uncommon for the meaning of the passage quoted to be quite different when the context is considered.

(4) He is allowed the use of notes; but only to *refresh his memory*. Such notes must have been made at the time of, or shortly after, the occurrence of the event, and must have been made by himself, or reviewed by him at the time if made by another.

(5) Every question must be answered by the witness, unless the answer would tend in any way to incriminate himself. If for reasons of professional secrecy he thinks a question should not be answered, he should appeal to the judge. The law in Great Britain does not recognize professional "privilege," and the secrets of patients obtained by medical men in the course of their profession cannot be withheld from the court. Nevertheless, where professional secrets are involved the witness should ask for the ruling of the judge.

(6) Last, but not least important, the witness should be well prepared on all parts of the subject on which he is to give evidence.

A. ALLISON.

MEDITERRANEAN FEVER (see UNDU-
LANT FEVER).

**MEDULLA OBLONGATA, LESIONS
OF** (see NERVOUS SYSTEM, CENTRAL, LOCAL
LESIONS OF).

MEGRIM (see MIGRAINE).

MEIBOMIAN CYSTS (*see* EYELIDS, AFFECTIONS OF).

MELÆNA (*syn.* Tarry Stools).—Melæna usually denotes hæmorrhage into the upper part of the alimentary canal. Though its source is commonly above the ileo-cæcal junction, no hard-and-fast line can be drawn, for considerable hæmorrhage into the lumen of the small intestine, if peristalsis is active or enhanced, may appear as bright-red blood in the stools, whilst small colonic hæmorrhages may be retained long enough for true melæna to occur.

Etiology.—(1) The blood may have been swallowed and may represent an epistaxis or an hæmoptysis, or may proceed from a bitten tongue in epilepsy, or a weeping aneurysm or other source of bleeding in the mouth, nose, or pharynx, or œsophagus. (2) Simple gastric or duodenal ulceration, gastrostaxis, and œsophageal hæmorrhoids resulting from portal obstruction, more particularly from cirrhosis of the liver, are important causes. The other gastric varieties of hæmatemesis (q.v.) seldom produce sufficient bleeding to alter the colour of the stools, though it may be found by chemical and spectroscopic examination—occult blood. (3) Lower down the small intestine the melæna may arise from tuberculous, typhoid, or malignant ulceration. The bleeding which accompanies intussusception seldom causes melæna, the blood being generally bright in colour, apart from the stools, and mixed with mucus. Some blood may be passed in severe enteritis, especially that produced by irritant poisons, but here it is hurried rapidly through the bowel and is generally bright-red in colour when evacuated. Ankylostomiasis may cause a protracted and continuous melæna, but is rare in the British Isles. Dysentery, though primarily productive of ulceration in the large bowel, may be associated with considerable oozing of blood into the stomach and small intestine. (4) Melæna may be a symptom of hæmophilia, leukemia, scurvy, purpura, toxæmic and infective jaundice, and other general conditions in which bleeding from mucous membrane occurs.

Bright-red blood is less mixed with the stools. When considerable, its chief sources are hæmorrhoids, rectal polypus, and ulceration of the colon or rectum, whether dysenteric, malignant, or stercoral, or due to non-dysenteric ulcerative colitis. It is well to remember that tuberculous and typhoid ulceration sometimes occur in the large bowel even more markedly

than in the small intestine. Lesser bleedings may be due to anal fissure, proctitis, thread-worms, mucous colitis, or constipation.

Diagnosis.—While the passage of bright-red blood is readily detected, the tarry stools of melæna may be simulated by those of patients taking bismuth, iron, or charcoal. The tarry stool of hæmorrhage has often a purplish tinge; that of bismuth, iron, or charcoal, a greyish or slaty colour. After iron, the stools only become black when they have been exposed to the air. Chemical and spectroscopic tests establish the diagnosis, and are also necessary to detect the passage of small quantities unrecognizable by the unaided eye—occult blood. (*See* FÆCES, EXAMINATION OF.) It must not be forgotten that blood appears in detectable amounts in the fæces of patients taking raw-meat juice and other hæmoglobin-containing foods.

Treatment.—This is essentially that of the cause, and will be found under the appropriate headings.

FREDERICK LANGMEAD.

MELÆNA NEONATORUM (*syn.* Morbus Maculosus; Hæmorrhagic Disease of Infants).—An uncommon disease of new-born children, in which constitutional symptoms are accompanied by hæmorrhage from mucous membranes. The etiology is obscure. Male infants are affected about twice as often as female.

Symptomatology.—The onset of the bleeding is usually on the third day, but the disease may date from birth or be postponed for a week or more. The blood appears first in the motions in about 50 per cent. of the cases, either mixed with the meconium or as small clots; soon it is passed unmixed, either fluid or as masses of clot. Hæmatemesis may occur alone, though usually in association with melæna. Other sources of bleeding are the mouth, nose, and navel, and there may be hæmorrhages also into serous spaces or the viscera, especially the suprarenal bodies. At first the temperature is usually raised. The child soon becomes profoundly shocked, with subnormal temperature, pallor, cold and blue extremities, quick feeble pulse, and suppression of urine; if untreated the disease often terminates fatally within three days of its onset.

Diagnosis.—Other causes of bleeding during the first weeks of life must be considered. *Septicæmia* is frequently confused with morbus maculosus, and can only be excluded definitely by a bacteriological examination during life. Sepsis, however, does not cause bleeding within

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a few hours of birth; examination of the navel may reveal its presence. *Syphilis* may be suggested by the family history, and detected by the Wassermann reaction. *Jaundice*, if severe, is accompanied by hæmorrhages at this age; slight jaundice has occasionally been noted in morbus maculosus. The melæna may be *spurious*, and be occasioned by swallowing blood arising from ulcerative stomatitis or syphilitic rhinitis, or ingested with the milk obtained from a breast with a cracked nipple. *Hæmophilia* is so very rare a cause of bleeding at this age as to be almost unknown.

Pathology.—Apart from the hæmorrhages, there are no post-mortem findings in most cases. In a few, punched-out ulcers have been seen in the œsophagus, stomach, and duodenum; though thought by some to be the source of the hæmorrhage, they are more probably due to necrosis following a sub-mucous extravasation of blood.

Treatment.—The modern treatment by subcutaneous injection of normal human serum has proved remarkably successful. The blood is withdrawn from the median basilic or median cephalic vein of the donor into a sterile flask, and the serum allowed to separate. It is then ready for use, and 10 c.c. or more may be injected twice or thrice daily. Transfusion, either by the direct or by the citration method, has given equally good results, but is more difficult to perform. Failing human serum, horse-serum may be tried. Gelatin 1 dr. of a 5-per-cent. solution may be given hourly by the mouth, or 2 dr. of a sterile 10-per-cent. solution may be injected hypodermically two or three times daily.

FREDERICK LANGMEAD.

MELANCHOLIA.—It is inevitable that at some period in the life of everyone a time of sadness and grief, caused by painful events, will arise. The sufferer is then melancholy, and naturally so. Melancholia differs from melancholy in that in many cases no cause for the condition is apparent. When there is an adequate cause, the state of depression is more intense and lasts longer than normally, and is less influenced by the environment. Melancholia may therefore be defined as an abnormal state of misery. It accounts for 20 per cent. of admissions into mental hospitals. The true frequency, however, is in excess of this figure, as many slight cases are treated at home and therefore do not appear in official statistics.

Etiology.—Hereditry of nervous or mental disease is traceable in a large proportion of

the cases; some observers record 50 per cent. The events which precipitate the attacks are mental shocks—for example, the deaths of relatives, or financial losses. Long-continued strains, such as nursing a sick relative, or ill-health, often end in this disorder. Insanity appearing in the period of lactation, and that associated with pulmonary tuberculosis, are frequently melancholic in character. One of the principal causes is old age, and this is so much the case that the followers of Kraepelin confine the use of the term melancholia to senile melancholia. The remaining cases are then included in manic-depressive insanity (q.v.).

Pathology and morbid anatomy.—Slight atrophy of the convolutions, with a compensatory increase in the quantity of the cerebro-spinal fluid, is present in a number of cases. On microscopic examination many of the nerve-cells may be found in severe cases to be degenerate, with defective staining of the Nissl bodies and displacement of the nucleus. It is doubtful, however, if these changes are in excess of those found in the brain of persons dying from other diseases. The true cause of the disease has yet to be discovered, and a long discussion on the subject would be out of place here. The theory most widely held is that a toxin circulating in the blood-stream and poisoning the cells of the cortex is responsible for the symptoms. The periodicity of the attacks in many cases, the occasional leucocytosis, and the presence of melancholia in admittedly toxic states, such as alcoholism, paralytic dementia, phthisis, and other diseases, all point in this direction. The nature of the toxin is unknown. A fact which tends to support this view is that the symptoms improve when constipation is relieved. Some would say, therefore, that it is derived from the intestinal tract. Others look for the poison in disordered glandular secretion.

Symptomatology.—The outstanding feature is the sad emotional tone. There is mental pain, which in some cases may be slight, but in others is so great as to keep the patient in continuous agitation, with wringing of hands, lamentations, and other signs of grief. The greater number of the patients, however, are dull, and take little interest in their surroundings, so absorbed are they in their own unhappy thoughts. When asked simple questions the response is slow, and at times a long interval elapses between question and answer. If an attempt is made to initiate a conversation the result is usually a failure and ends in mono-

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syllabic answers to questions. This is one of the results of a feeling of resistance in the environment which many of these patients have. There is a sense of impotence to use the brain, and just as a severe blow on a nerve trunk may paralyse a limb, so severe mental pain may paralyse the power to think, feel, and act. And in this connexion it is bad practice to tell the patient to rouse himself. This is what he wants to do, but cannot (Maudsley). The patient has often a considerable amount of insight into the condition, and realizes he is in an abnormal state. In addition to the feeling of misery, there is frequently present a state of anxiety which the patient, when he argues out the matter, may see to be ungrounded, yet is unable to shake off. Loss of natural affection towards family or friends causes great distress. In uncomplicated cases memory is good for events prior to the illness, but not for events that have occurred during it, as attention, one of the necessary factors, is always defective.

Physical signs.—The patient looks thin and ill, and there is loss of weight. The expression of face is characteristically sad. The forehead is wrinkled, with transverse furrows in the middle, flanked by vertical ones at the inner ends of the eyebrows. This arrangement has been compared to a horseshoe, or the Greek letter Omega. The inner ends of the eyebrows are raised, and the corners of the mouth droop. Darwin has pointed out the similarity of this expression to that found in the crying child. Temperature is subnormal. The tongue is coated and the breath foul. There is deficiency in the amount of pepsin secreted, and constipation is almost invariably present. The pulse tension is stated to be higher than normal, but it is doubtful if this is the case. Anæmia is frequently present. Respiration is shallow and slow. The urine is decreased and the specific gravity increased. Males are impotent, and in females menstruation is in abeyance. The skin is dry, the nails are brittle and inclined to split, the complexion is muddy, the hair grey. The chief nervous disturbances are those of the motor functions. The attitude is characteristic, and resembles that of paralysis agitans. The head and trunk are inclined forwards, with slight flexion of hips and knees. There is flexion at the shoulder-joints, and the elbows are at right angles and held rigidly to the sides. There is proximal rigidity—that is, the muscles round the large joints are contracted, and when movement occurs it is

chiefly at the peripheral joints. For example, in agitated melancholia the fingers are in constant movement. These patients shake hands from the wrist, and walk slowly and stiffly, more from the knees than the hips (Stoddart). Speech is slow and indistinct. Writing also is slow. The sensory system is little affected, though many patients complain of a peculiar sinking feeling in the epigastrium. The knee-jerks are brisk, with quick return, and the plantar reflexes are flexor in character.

The foregoing description includes the prominent symptoms and physical signs of melancholia. There are several other features common to melancholia and other mental diseases, though some are most frequently met with in the former condition.

Sleep.—This is always disordered, especially at the beginning of the illness. The number of hours slept should be recorded upon a chart, and this is often a valuable help in estimating the progress towards recovery.

Resistiveness.—Many patients are stubborn, and resist everything that is done for them; they resist being dressed, washed, etc. This resistiveness is probably made up of two factors—real opposition, and the rigidity noted above. Under this heading may be included refusal of food, which may be due to stubbornness, or may be the result of a delusion, or of a systematic attempt to end life.

Suicide.—Every melancholiac is a potential suicide, and this is the great danger of the disease. He is so because of the not altogether unreasonable desire to end a state of misery. In other cases the patient is suicidal because of a delusion, for example, that his death is necessary to the salvation of the world, or because hallucinatory voices urge him to take the step. Or a man who has the delusion that people are following him to kill him may, in order to save himself from his imaginary pursuers, jump out of a window and be killed.

Bad habits, such as biting the nails, picking sores in the fingers or the face, are generally thought to be of ill omen, though I have known cases with these symptoms recover. The patients, as a rule, are cleanly in their habits, though less careful about their appearance than when in health.

Varieties.—Melancholia may be divided into (1) simple melancholia, (2) delusional (including hypochondriacal) melancholia, (3) agitated melancholia.

1. **Simple melancholia** includes the case

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with depression unaccompanied by delusions, hallucinations, or agitation.

2. Delusional melancholia.—In this condition the patient attempts to account for his misery, and the explanation is a delusion. Such delusions may be of all kinds. One which is frequently met with is the belief that all hope of salvation has been lost owing to general unworthiness, or wickedness, or because "the unpardonable sin" has been committed. Petty misdeeds years old are magnified into crimes for which there can be no forgiveness. This is the "religious mania" which is so frequently spoken of by the laity, but it is a term which is of no value, and should therefore be avoided by medical men. In other cases the delusions are constellated in a persecutory system. The patient is persecuted by being followed about, insulted, or tortured by electricity, X-rays, radium, wireless telegraphy, or any other phenomenon which is the scientific curiosity of the day. Hallucinations may be of hearing, of sight, of taste, of smell, or of cutaneous sensations, but the auditory are the most frequent. Hallucinations often form the starting-point for the delusions. For example, the patient, owing to hallucinations of smell, may have the delusion that noxious gases are being blown upon him through the keyhole. When the delusions refer to the general health the case is one of hypochondriacal melancholia. Here the complaint will be expressed that the *prima via* is blocked, that the bowels never act, or that a tumour is growing, etc.

3. Agitated melancholia.—The patient, in place of being, so to speak, stupefied by his grief, is in continuous movement, pacing up and down the ward, wringing his hands, moaning, and calling on all to witness his distress.

Course.—The attack as a rule develops gradually and as gradually declines, the duration being about eight to fourteen months. After recovery the patient may keep well for the remainder of his life. On the other hand, relapses frequently occur, and then the case is one of recurrent melancholia. In one of the recurrences mania may take the place of melancholia, and the case is one of alternating insanity. (See MANIC-DEPRESSIVE INSANITY.)

Diagnosis.—This rarely presents any difficulty. Many cases of melancholia are called hysteria or neurasthenia, more as a sop to please the relatives than from clinical observation. In every mental disorder it is necessary to exclude *general paralysis of the insane*, and

it must be remembered that in not a few cases of this disease the prominent mental symptoms are those of melancholia. In general paralysis the characteristic physical signs can almost certainly be found, as changes in the pupils, speech defects, tremors, ataxy, or altered reflexes. If there is doubt, an examination of the blood and of the cerebro-spinal fluid will settle the question, though it would be absurd to suggest this in every case of melancholia in the fear that the graver disease might be present. *Dementia præcox* in an early stage is at times difficult to exclude, and in some cases the diagnosis can be made only by waiting; rigidity, resistiveness, and periods of stupor are common to both diseases. In *dementia præcox*, though the prevailing tone at the time of the examination may be sadness, yet probably there will be some signs of the emotional deterioration characteristic of the disease. Visits of friends, receipt of letters, and other emotional factors may make the melancholiac worse, while the patient with *dementia præcox* is unaffected and indifferent. There are, moreover, few cases of *dementia præcox* in which some of the characteristic mannerisms, the stereotypy, or the hypersuggestibility will not, on careful examination, be found. *Anxiety neurosis* as described by Freud is characterized by general irritability, anxious expectation, and physical symptoms such as palpitation, dyspnoea, attacks of perspiration, tremor, diarrhoea, and vertigo. In the majority of cases of melancholia misery is more prominent than anxiety, and the physical disorders are absent.

Prognosis.—The termination of simple melancholia is as a rule in recovery. A few cases become chronic, yet even these may eventually recover. A small percentage of the patients die, wasting away despite all efforts to keep them alive. Signs suggestive of a favourable result are the return of sleep, of appetite, of an interest in surroundings and in occupation. Increase in weight is also of good import. Unfavourable signs are the development of persistent hallucinations and delusions, long-continued refusal of food, the formation of bad habits such as picking of the fingers, and deterioration of memory. A case of hypochondriacal melancholia rarely recovers. Too sudden a recovery reveals a mental instability which is of bad omen.

Treatment. General measures.—The first question to be decided in every case of mental disease is whether institutional treatment is

necessary or not. While in most cases there can be no doubt that the patient will have the best chance if sent away from his relatives, yet, as this involves certification with its accompanying stigma, it is not always possible to recommend it. Expense has to be considered, and home treatment is costly if sufficient nurses are engaged. For the purpose of this article, however, it will be assumed that the necessary funds are available, and that the practitioner can treat the patient at home. The danger of suicide must be kept ever in mind. To lessen the risks, the patient must live day and night on the ground floor, and the windows be prevented from opening more than 5 in. at the top and bottom. All articles such as scissors, razors, knives, and pieces of string must be removed, and all medicine bottles and antiseptics locked up. As the open fire may be a danger, it may be necessary to provide a suitable locked guard. But the safety of the patient will be mainly in the hands of the nurses, and to these definite orders must be given that he is not to be allowed out of sight for a moment, not even when the calls of nature are attended to. The fact that the patient expresses himself as afraid to die or thinks suicide wicked must not throw one off one's guard, as an impulse may arise which he is powerless to resist. He must be kept in bed, at any rate at the beginning of the illness; and if it is possible to provide a small veranda on which he can have open-air treatment, so much the better. As he improves, exercise may be ordered, but it must be carefully regulated. Baths are useful, the prolonged warm bath in cases of agitation, and the cold shower or electric bath if the case threatens to become chronic.

Diet.—The digestion is disordered, and light and easily digestible food must be given. What Clouston calls the "gospel of fatness" should be remembered, and plenty of milk and cream, cod-liver oil, etc., provided. If the patient refuses all nourishment, he must be forcibly fed. Rectal feeding is of little value, though large saline enemata are at times of service. Often a tactful nurse can overcome the resistance of the patient, and he can be spoon-fed. If this is of no avail, and the subject is strong and healthy, he is placed on his back, firmly held, and small quantities of milk poured by means of a feeding-cup into the cheek. This may be swallowed if the nose is closed, but an artful and perverse patient can eject it from the mouth, and, if so, there must

be recourse to tube-feeding; particulars of this are given in the article **INSANITY, TREATMENT OF**. It is important to see that a sufficient amount of water is taken, and to insist that everything the patient swallows is noted in writing by the nurse. The weighing-machine should be frequently in use; it is of more value than the clinical thermometer.

Medicinal treatment.—Drugs are used chiefly to relieve symptoms. Constipation is often obstinate, and has to be met by appropriate remedies. Larger doses may be necessary than those required for sane patients; the nurse must pay special attention to this matter, as the statements of the patient may be misleading. It is advisable to give some simple digestive stimulant, such as a mixture containing bicarbonate of soda, nux vomica, and gentian, before meals. If this is found unsuitable, an acid mixture containing pepsin, after meals, is often of value, bearing in mind the deficiency of pepsin in the gastric juice. As convalescence becomes established, tonics containing iron and arsenic will assist recovery. Sleeplessness will require treatment. The rule is to use hypnotics as little as possible. Sleeplessness is often a bad habit, and, should the habit be broken, natural sleep may come. Therefore, use an hypnotic for one or two nights in sufficient dosage and stop. The next question will be, which is the best hypnotic. There is a large list to choose from: paraldehyde, sulphonal, trional, veronal, amylene hydrate, the bromides, and a host of others. No definite rule can be given, and each may have to be tried in turn till the one most satisfactory to the patient is discovered. In slight cases the bromides may suffice. Paraldehyde is a safe but disagreeable drug. Veronal has secured a firm footing in the list of trusted hypnotics. Dial, a newer drug, is in many respects very satisfactory. The combination of two hypnotics often acts better than either singly. Opium is rarely of service in the treatment of mental disorders, but in some cases of melancholia it does well. Begin with small doses, increase slowly to large ones, and then gradually diminish again. The administration of thyroid gland in large doses has been found beneficial in some cases of stupor. Psycho-analysis has been tried in a few cases, and success has been recorded. Hypnotism is stated to have been used with beneficial results. The fact that many patients recover without recourse to such psycho-therapeutic measures naturally gives rise to doubts as to their efficacy.

R. H. STEEN.

MEMORY, DISTURBANCES OF

MELANURIA (*see* URINE, EXAMINATION OF).

MEMBRANA TYMPANI, INFLAMMATION OF (*see* OTITIS MEDIA).

MEMBRANOUS COLITIS (*see* COLITIS).

MEMBRANOUS CROUP (*see* DIPHTHERIA).

MEMORY, DISTURBANCES OF.—Disturbances of memory may be divided into three main types—*hypermnnesia*, in which the memory is abnormally acute; *amnesia*, in which it is defective or lost; and *paramnesia*, in which it is distorted. Each of these three groups will be separately considered.

Hypermnnesia is frequently observed in the hypnotic state, and occasionally occurs spontaneously in certain pathological conditions. It has little clinical importance.

Amnesia is a common phenomenon met with in a great number of disorders, but of very variable origin and significance, according to the nature of the case in which it occurs. Memory is not a simple process, but really comprises three distinct processes, and a failure of memory may arise from a defect in any one of the three. These three constituent processes are *impression*, whereby an event is impressed upon the mind, *retention*, whereby the impression is conserved in the mind, and *recall*, whereby the retained impression is brought again into consciousness when the necessary conditions are present. If any of the three essential parts of the machinery of memory is disturbed, an amnesia will result, but its significance cannot be understood until the precise process disturbed and the nature of the disturbance have been elucidated.

Disturbances of impression. If the state of mind present when an event occurs is such that the event is not recorded, an amnesia due to a failure of impression results. The most notable examples of this type are found in the psychoses, particularly in the confusional insanities. Here the occurrence of events is obviously not observed and registered by the patient, and hence no subsequent memory of them can exist. A similar condition obtains in febrile delirium. The emotional apathy characteristic of dementia præcox, again, sometimes leads to a defective memory because the patient's interest is withdrawn from the external world, and hence what occurs therein makes no impression upon him. A failure of memory due to a closely similar mechanism is often observed in the psychoneuroses. The patient's attention is distracted by various preoccupations and

anxieties, events are not therefore observed sufficiently to be recorded, and an apparent lack of memory results, which is frequently a source of further anxiety and apprehension to the patient. This fact is of considerable clinical importance, because it is necessary to explain what is taking place to the patient, and to reassure him that his apprehensions that he is losing his memory have no real foundation.

Disturbances of retention.—Here the disturbance of memory is due to the fact that, although the event is duly observed and recorded, the resulting impression is not retained. Amnesias of this type occur almost exclusively in cases of organic origin, notably in the organic dementias. They are found, for example, in senile dementia, general paralysis, and alcoholic dementia. In the last, the defect of retention may be so pronounced that events are forgotten almost immediately after they have happened, so that, although the patient is quite able to understand and appreciate what is taking place around him, memory for these occurrences only persists for a few moments and is then completely effaced. This phenomenon is sometimes termed "*instantaneous amnesia*." It inevitably carries with it an inability on the part of the patient to orientate himself adequately either in space or time. Amnesias due to a failure of retention generally obey the so-called "*law of regression of memory*," memories being lost in the inverse order to that in which they have been acquired, so that recent happenings are rapidly forgotten while those of remoter date are comparatively well preserved. This phenomenon is, of course, met with in normal senility, and partly accounts for the tendency of the old man to live in the past, but it attains its most extreme degree in senile dementia. Patients suffering from this disorder are frequently unable to remember any of the events of the preceding day or even hour, while they are quite capable of giving an accurate account of their school days and early life.

Disturbances of recall.—In this group events are duly recorded in the mind, and the impressions are retained, but the corresponding memories cannot be recalled. In other words, the memories exist in the mind but they are not available. Minor disturbances of this kind are frequently observed under normal conditions, for example, when we are temporarily unable to recall some name with which we are perfectly well acquainted. The more pronounced degrees of amnesia due to failure of recall,

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however, occur mainly in the psychoneuroses, and especially in hysteria. Here the amnesia often relates to definite sections of the patient's past experience, so that he is unable to remember any of the events which have occurred during a certain sharply limited period. This phenomenon was frequently observed in the war hysterics. A patient, for example, might exhibit a gap in his memory extending from a certain day when he was perhaps temporarily stunned by the bursting of a shell, to a day a month later when he found himself in a base hospital with no recollection of anything that had transpired during the preceding month. That the amnesia in such cases is not due to any disturbance of impression or retention, and that the memories still actually exist in the mind, is demonstrated by the fact that recollection for the whole period in question can be restored by the use of hypnosis or other psychological methods, or may subsequently return spontaneously. The amnesias which follow hysterical somnambulism and fugues are all of this type, as are also the reciprocal amnesias existing between the different personalities in cases of double personality. Similar amnesias are sometimes observed in certain psychoses, and they can be produced artificially by hypnosis.

The so-called "system amnesias" are uncommon phenomena occasionally occurring in hysteria. Here the loss of memory does not comprise any particular period of time, but relates only to certain systems of ideas. Thus a patient normally able to converse fluently in several languages may suddenly lose command of one of these languages entirely, and become absolutely unable either to understand or to speak it.

The psychological mechanism responsible for the majority of amnesias due to lesions of recall consists in the formation of a dissociation of consciousness. A portion of consciousness is, as it were, split off from the main body, so that its contents are no longer directly accessible. This dissociation can be overcome by the employment of various psychotherapeutic methods, and the missing memories thereby restored to consciousness.

A false or pseudo-amnesia is often observed in neurasthenia and psychasthenia, and it is important to distinguish this phenomenon from the actual amnesias hitherto described. In cases of this type the patient complains that his memory is failing, and that he feels himself increasingly unable to recollect current

events, the details necessary for his business, and so forth. An objective test, however, establishes the fact that the patient's memory is nearly as good as it ever was, and at any rate vastly better than he believes it to be. Such defect as actually exists is to be ascribed to the inattention and preoccupation with internal anxieties already described under "lesions of impression." Apart from this the amnesia is only apparent, or, more accurately, it is merely a subjective feeling that memory is impaired. It may, indeed, be laid down as a rough clinical rule that actual impairment of memory and subjective appreciation of impairment tend to vary inversely. That is to say, a patient with actual severe defect of memory rarely complains of it, while in a patient who complains of defect of memory the actual defect is rarely serious.

Paramnesia.—Here memory is distorted, so that recollections which appear to the patient as genuine are in fact perverted or wholly fictitious representations of the real past. Paramnesia necessarily involves a certain amount of amnesia, because the presence of fictitious memories must naturally be accompanied by at least a partial forgetting of the events which actually took place at the time in question. The most striking examples of paramnesia are found in the *confabulations* characteristic of certain alcoholic insanities, notably in Korsakoff's or polyneuritic psychosis. Here the patient, while actually unable to recollect any of the real events of his recent past, furnishes a complete and detailed account of his doings which investigation shows to be entirely fictitious. An interesting feature of these fictitious memories is that they are easily influenced by suggestion, so that the patient can be made to give any history that is desired provided that appropriate leading questions are used to elicit it. Similar confabulations are also sometimes observed in the senile psychoses.

Paramnesia is frequently present in delusional states, where the memory of the past is distorted by the delusional system. A patient who believes himself to be the king will, for example, describe his early life in the royal palace, the wars he has conducted, and so forth.

Confabulation also constitutes the characteristic feature of *pseudologia phantastica*, a condition in which the patient, without any other notable mental symptoms, is constantly relating elaborate histories of events he has

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figured in, which are found to have no foundation whatever in fact. The events described are generally of a kind which exhibit the patient playing an interesting or distinguished part, and which obviously minister to his feeling of self-importance. This also holds of the closely akin confabulation not uncommonly observed in hysteria. In all these conditions the psychological mechanism is obviously nearly allied to that responsible for the "day-dreaming" of adolescence, except that instead of being regarded merely as pleasant figments of the imagination the phantasies are projected as memories of actual occurrences.

Finally, it must be noted that, although the phenomena just described are apparently so bizarre, minor instances of the same essential psychological process are frequently observed in everyday life. It has been experimentally shown, indeed, that a large number of our memories contain definitely paramnesic elements. Our memories of the past are often gilded by our emotions, so that we tend to recollect our past exploits in a way which places them in an unduly favourable light. In times of great emotional stress, distortion of memory may be considerable; and the detailed description of entirely imaginary events given by perfectly honest people at the time of the famous "Russian rumour" provided a convincing demonstration of the extraordinary extent to which this may occur. It is important that the practitioner should constantly bear in mind the existence of this psychological process, especially in dealing with neurotic patients, and that he should clearly realize that between accurate memory and deliberate lying there is a whole series of gradations where the past is distinctly but unconsciously falsified.

BERNARD HART.

MENIÈRE'S DISEASE. — A symptom-complex more common than was formerly supposed, the essential symptom of which is dizziness, or vertigo; tinnitus and defects in hearing are usually but not always noticed. The disturbances are due to various causes which affect one or other part of the vestibular system.

The attacks of vertigo may be mild, or very severe, and accompanied by bulbar reactions, such as nausea, vomiting, diarrhoea, vasomotor disturbances—pallor, or flushing and sweating, with cardio-vascular symptoms—faintness and dyspnoea. Staggering is sometimes marked, or outstanding apart from shock

symptoms. Occasionally there occurs violent titubation, the patient being suddenly hurled to the ground without any warning sense of dizziness and without paralysis or loss of consciousness. Frequent recurrence of even mild attacks in one form or other is liable to produce psychopathic disturbances, including manifestations of hysteria and hypochondriasis which are apt to lead the practitioner away from the physical cause. True epileptic seizures may occur.

Subjects of recurrent attacks of vertigo can generally discover no assignable cause, though stooping and tight neckwear have been noticed to bring them on in some people. They may take place during day or night, when lying down or getting up, when resting quietly or taking exercise, during meals or while writing or reading. Diet seems to play no important part. Because it has been noticed that the majority of patients with Menière's disease are the subjects of postnasal catarrh and Eustachian obstruction, attention has been focused on the correlation of this disease and the efficiency of the Eustachian tube. It has been found that this is no independent coincidence. In the first attack patients do not complain of tinnitus or deafness, and attribute the giddiness to being "out of sorts," to an "influenzal cold," or to "gastric troubles" or "biliousness" or "a touch of liver"—especially so when vomiting and nausea are prominent. When faintness and collapse occur the heart is liable to be blamed, and in these various ways it happens that the state of the Eustachian tubes is often disregarded. Deafness may antecede or accompany an attack and may pass off or increase. Tinnitus, though common both before and after an attack, is not invariable. Nystagmus can often be observed when sought for, especially during a seizure accompanied by staggering; it is usually rotatory or mixed and asymmetrical; occasionally it is vertical or horizontal. The ocular symptoms naturally lead to investigations for visual defects, but though there is a close connexion between visual and vestibular disturbances, the correction of an error of refraction cannot be expected to cure the ordinary form of Menière's disease.

Lesions in Menière's disease.—Although the first fatal case recorded by Menière, in the middle of last century, appeared to him to be due to hæmorrhage into the semicircular canals, it is now claimed that the case was one of *infective labyrinthitis* which terminated in

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meningitis serosa; for the hypervascularity in membranous labyrinthitis certainly resembles free hæmorrhage in the semicircular canal to the naked eye, while histological and bacteriological examinations are necessary to reveal the true nature of the disease. Moreover, meningitis serosa, which may follow labyrinthitis, does not leave signs which would have been recognized in Menière's time; it nevertheless explains the coma which occurred in his case and in parallel cases seen at the present time. Acute inflammation of the labyrinth is only one form of Menière's disease, and occurs as a complication of acute or chronic otitis media. Proved cases of hæmorrhage into the labyrinth are extremely rare; they occur occasionally in the course of definite disease of the vascular system, e.g. leucocythemia (Mott and Lake). The symptom-complex occurs also in the course of formation of brain tumours, particularly auditory-nerve tumours, and in certain diseases of the cerebral vessels, notably of the basilar artery.

The labyrinth tests described under EAR, EXAMINATION OF, are of the highest value in the differential diagnosis of Menière's disease. Generally speaking, when there is labyrinthitis the labyrinth becomes irresponsive to the caloric and rotation tests, and also to the galvanic tests in cases of auditory-nerve tumour.

Injuries of the head and temporal bone (e.g. war injuries) are often followed for some weeks by Menière's symptom-complex, due to damage to the vestibular systems. Another cause is syphilis, which is associated with signs of lesion of the vestibular and cochlear divisions of the auditory nerve, with or without facial paralysis. *Bell's palsy*, without involvement of the eighth nerve, may be accompanied by vertigo, possibly due to tremor or paralysis of the stapedius, and consequent unequal labyrinthine pressures.

Symptoms of Menière's disease can be provoked by changes in barometric pressure, and are met with in caisson divers, aviators, and mountaineers, especially during rapid descents, which are accompanied by compression phenomena. Comparable are descents in funicular railways of a thousand feet or more, and in very deep mine shafts. In these cases the vertigo is not due to hæmorrhage into the labyrinth, even though there may be hæmorrhage from the drum of the ear owing to too rapid compression or decompression; the cause of the vertigo is an inequality of pressure

in the labyrinth, induced by inefficiency of the Eustachian tube on one side.

The most frequent form of Menière's disease seen in ordinary clinical practice is similarly due to unilateral inefficiency of the Eustachian tube with consequent pressure disturbances in the intact labyrinth. In these cases the labyrinths respond to all the tests applied: (1) rotation, (2) caloric, (3) galvanic—and there is consequently no evidence of disease within the labyrinth or vestibular nerve. There may be no deafness, in fact there is sometimes hyperacusis for low (basal) tones.

It must be emphasized that the Eustachian inefficiency is liable to be overlooked in the first instance; it may then lead to Eustachian catarrh, leaving the patient with more or less deafness and tinnitus.

Treatment. We must first ascertain and deal with the cause (see OTITIS MEDIA). When the trouble is associated not with otitis media but with colds and catarrhs, attention must be directed to the local disease, be it rhinitis, pharyngitis, adenoids, or tonsillitis. The most frequent form of attack—that due to Eustachian obstruction—can be cut short by successfully re-establishing and maintaining the efficiency of both tubes. This is usually the difficulty in general practice. A choked or congested tube may require repeated inflation by Valsalva's method or by politizerization, or the catheter may be the only certain means of giving immediate relief. Attempts to self-inflate should be made with the face downwards; lateral flexion of the neck during the act sometimes ensures success. Some patients discover this method for themselves; instinctively they assume a resting posture, as head-movements intensify the giddiness; an upright sitting position with a head-rest, such as in a grandfather's chair, is often better than recumbency.

Drugs do not appear to be of much use, nevertheless adrenalin, inhalations, or chlorotone, by reducing local congestion of the mucosa, may give relief to the obstruction. Iodides achieved a repute perhaps through their tendency to promote secretion of more watery mucus, and because their use has been followed by cure in the occasional cases attributable to syphilis. Bromides may act as sedatives to an over-responsive labyrinth, or may possibly relieve spasm of intratympanic muscles. Quinine has been recommended as sedative in minute doses, but its beneficial action, if any, may conceivably be anti-catarrhal.

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In cases of obstinate Eustachian obstruction, fenestration of the drum-membrane is a rational expedient. Destruction of the labyrinth has been occasionally practised, but is now considered to be rarely, if ever, necessary in non-suppurative cases.

Local measures directed to the vestibular apparatus may have to be supplemented by general treatment, especially in patients who have been overtaxed by mental or physical fatigue, but local causes should never be overlooked or neglected. It is to be remembered that the discovery or removal of nasal obstruction and the cure of postnasal catarrh are at once logical and sound methods of effecting the cure of many cases of Menière's disease.

SYDNEY SCOTT.

MENINGEAL HÆMORRHAGE (see CEREBRAL VASCULAR DISEASE).

MENINGEAL HÆMORRHAGE, TRAUMATIC (see HEAD INJURIES).

MENINGITIS.—Meningitis is an inflammation of the membranes of the brain or spinal cord, due to their infection by micro-organisms. In all cases in which the pia-arachnoid is invaded the meningitis tends to generalize itself and become cerebro-spinal in distribution, but the name cerebro-spinal meningitis or fever is reserved for that form caused by the *Diplococcus intracellularis* of Weichselbaum (PLATE 4, Fig. 4, Vol. I, facing p. 146). This article will describe—

1. SEPTIC MENINGITIS.
2. SEROUS MENINGITIS.
3. EPIDEMIC CEREBRO-SPINAL MENINGITIS.
4. TUBERCULOUS MENINGITIS.

Syphilitic cerebral meningitis is considered under CEREBRO-SPINAL SYPHILIS.

1. SEPTIC MENINGITIS

Etiology.—The infecting organisms obtain access to the cranial cavity in several ways:

1. Most commonly from *pus foci in the neighbourhood of the brain*. (a) When the infection starts from an injury to the skull, the meningitis may be localized to the dura mater and thus cause a pachymeningitis externa or interna; in such cases the inflammation usually goes on to abscess-formation. The most dangerous kinds of injury to the skull are the punctured wounds, since they so readily escape detection. Infection may occur in compound fractures, or even without a fracture if the outer table of the

bone has been exposed. (b) By infection from purulent disease of the mucous membranes lining the cavities of the temporal bone or accessory nasal sinuses. (c) From any suppurative process of the scalp or face, e.g. carbuncle, erysipelas, or orbital inflammation.

2. Via the blood-stream from some distant site of suppuration. Most commonly the focus is in the lungs, as in bronchiectasis. In rarer cases infection is derived from pericarditis, infective endocarditis, liver abscess, dysentery, disease of the female pelvic organs, and septic disease of bones or joints.

3. More rarely meningitis results from direct invasion of the blood-stream in such general infections as typhoid fever, influenza, and pneumonia.

Pathology.—In injuries to the skull the pus-forming organisms may be conveyed directly from the infecting focus, but they often spread inwards along infected venous or lymph-channels. Disease of the middle ear is one of the most frequent and important sources; the micro-organisms gain access to the meninges along the walls of blood-vessels, or extend directly through the inflamed dura mater from the carious or necrotic bone; when the labyrinth is diseased they may spread along the nerve-sheaths and through the internal auditory meatus.

Morbid anatomy.—When the infection comes from disease of neighbouring structures the meningitis may be local, and limited to the base or convexity of the brain or to the spinal meninges. Occasionally the infection is disseminated, in other cases it is generalized over the whole cerebro-spinal axis. Inflammatory changes are also found in the lining membrane and choroid plexuses of the ventricles, and these may be distended with sero-purulent or purulent fluid. The extent of purulent deposit varies greatly. The pus formed by the streptococcus and staphylococcus is yellowish, and often diluted with inflammatory serous fluid. The vessels of the brain or cord are engorged, and the purulent exudate extends along their sheaths into the nervous tissues, producing lesions which, though minute, indicate that all leptomeningitis is also associated with some encephalitis and myelitis. Microscopically the usual inflammatory changes are found, and the causal agent can often be obtained.

Symptomatology.—The signs and symptoms of the localized forms of meningitis of the dura mater arising from injuries or diseases of the cranial bones are clinically related with

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abscess of the brain and are not considered here. (See CEREBRAL ABSCESS.)

The onset of *diffuse septic leptomeningitis* varies considerably, and may be more or less masked by the symptoms of the primary disease. The characteristic symptoms are general and special. The fever is rarely high, though it often reaches 103° F., and is usually intermittent; the pulse-rate is often very rapid, but not infrequently there is a disproportion between pulse-rate and temperature. The mind soon becomes clouded, and the patient passes into varying degrees of unconsciousness; delirium is frequently present and may be violent. Pain in the head is an early symptom; it is often severe, and even in partial unconsciousness the patient gives indications of it. Vomiting also occurs early, and may be very frequent. The bowels are at first constipated, but as coma deepens both urine and feces are voided involuntarily. The rate and character of the respirations are altered; the variations depend on the degree of intracranial pressure. The rate is often very rapid, but sometimes there are periods of alternating slowness and frequency—the “cerebral” type of respiration. Cheyne-Stokes rhythm may occur. The *special signs* depend on the site on which the maximum intensity of the inflammation falls. Convulsions are suggestive of irritation of the cortex. Rigidity and retraction of the neck are present when the meningitis invades the posterior fossa. The back and abdominal muscles are frequently in a state of varying and irregular spasm. Kernig's sign is often found, though it is not so marked as in epidemic cerebro-spinal meningitis. The reflexes vary considerably; at first brisk, they may at a later stage disappear. Sometimes the plantar response is of the extensor type. The cranial nerves are not greatly affected. The pupils gradually cease to act to light and become dilated. Optic neuritis is not often found unless complications exist, and if present is only of slight degree. The muscles of the eye and of the face are sometimes paralysed.

Diagnosis.—This must be carefully considered in the light of the etiology, and the possible masking of the meningeal signs by the primary disease must always be borne in mind. In purulent disease of the ear the possibility of abscess, sinus thrombosis, meningitis, or combinations of these conditions, is to be remembered. Abscesses of the temporo-sphenoidal lobe are frequently uncomplicated, and present the characteristic features of depressed tem-

perature and slow pulse. Abscesses of the cerebellum are, on the contrary, much more frequently complicated by other pyogenic intracranial lesions, and the diagnosis may be very difficult. In many such cases the diagnosis is based chiefly on the result of examination made by the surgeon during the performance of the radical mastoid operation, and on the findings of a lumbar puncture. The cerebro-spinal fluid in diffuse septic meningitis is turbid or even purulent, contains a large number of polynuclear leucocytes, the protein is in excess, the fluid does not reduce Fehling's solution, and its alkalinity is diminished. Pyogenic organisms may be found in it. Sometimes, however, the fluid may approach the normal, even though meningitis is present.

In ear disease in children the diagnosis may be especially difficult, for simple purulent otitis may give rise to grave cerebral symptoms such as fever, drowsiness, rigidity of the neck, alteration in the pupils, all of which may disappear on puncture of the tympanic membrane.

Prognosis and treatment.—Fully developed diffuse septic meningitis is a very fatal condition, but a certain proportion of the cases certainly recover. During the late War many men with gunshot wounds of the skull who developed generalized meningitis, and from whom turbid fluid containing living micro-organisms was obtained by lumbar puncture, made complete recoveries. A few cases of recovery have even been recorded in which the infection was streptococcal. If signs of intracranial mischief are present, prompt treatment of the original infection may locate the disease and remove the focus. Drainage of the infected meningeal space by frequently repeated lumbar puncture offers the best chance of recovery. Surgical drainage of the subarachnoid space through the foramen magnum or the posterior fossa of the skull has in rare cases been successful. Urotropine has been employed, but it is doubtful if it has any effect.

2. SEROUS MENINGITIS

Etiology.—There are a few conditions in which serous effusions within the meninges assume clinical importance. The cause is usually a septic focus outside the skull, generally in the immediate neighbourhood, as suppurative ear disease, but occasionally at a distance, as in the appendix or pelvic organs. If the organisms are of low virulence, they may be unable to invade the meningeal spaces, but their toxins cause a serous inflammatory re-

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action in the meninges, the ependyma of the ventricles, or in the brain tissue itself. The inflammation may not proceed beyond this stage, or may be arrested by removal of the pyogenic focus.

It is possible that the condition of "meningism," which occurs in many of the specific infections, is due to a serous meningitis. Its symptoms are commonly attributed to a congestion of the brain.

Apart from such cases, in which the microbic origin of the inflammation can be clearly discerned, there are met with, from time to time, others presenting grave cerebral symptoms in which, either at operation or at post-mortem examination, the only lesion found is an excess of fluid in the pia-arachnoid cisterns or in the ventricles of the brain. In these cases a history may sometimes be obtained of injury to the head, or of an obsolete focus of sepsis, as in the ear, or of previous cerebral attacks dating back to childhood. Quincke, who first directed attention to this condition, described a number of cases, chiefly in young women, which set in with sudden and severe cerebral symptoms, as headache, vomiting, and optic neuritis. No very definite cause could be ascertained. Quincke considered the attacks to be in the nature of angio-neurotic oedema. Sometimes, in the aged, death is due to cerebral symptoms which end in coma, and the only lesion found is an excess of the clear fluid within the cranium. This condition is called *simple or serous apoplexy*. A similar exudation is present in some fatal cases of sunstroke.

Clinically, the cases may be classified into two groups, the one being acute or subacute in onset, resembling the more fulminating forms of intracranial disease; the other slower in evolution, but liable to exacerbations and producing more the clinical features of cerebral tumour.

Diagnosis.—In many cases the diagnosis cannot be made with certainty. It should be considered when symptoms of increased intracranial pressure are present and the conditions that generally produce it can be excluded. The cerebro-spinal fluid withdrawn by lumbar puncture is clear, sterile, does not contain excess of cells, but is under pressure and may show some slight excess of protein.

Treatment should be directed in the first place to removing any focus of infection. In many cases the symptoms subside quickly after a radical operation on the ear, if there be disease there. Repeated lumbar puncture

is strongly recommended, and may produce complete recovery.

3. EPIDEMIC CEREBRO-SPINAL MENINGITIS (*syn.* Spotted Fever, Malignant Purpuric Fever, "The Black Death")

An endemic disease, of which epidemics of varying severity occur. The chief factors in these outbreaks is the human carrier, who himself appears to acquire immunity to the disease. The predisposing factors are overcrowding, fatigue, and debilitating infections such as coryza, influenza, and certain of the specific fevers. By far the greater number of cases occur in children from 4 months to 5 years of age, and in Great Britain at least the disease is most rife in the early months of the year. The causative micro-organism is the *Diplococcus intracellularis* of Weichselbaum. There are several forms of this (as Gordon's types I, II, III, and IV) and the different manifestations of the disease have been attributed to infection by these different types. The most probable paths of infection are (1) from the naso-pharynx via the lymphatics, (2) along the lymphatics in the spinal nerves, (3) by the blood-stream from the naso-pharynx or accessory sinuses. The incubation period is from four to ten days.

Symptomatology.—The general symptoms resemble those of other forms of acute infective meningitis. The nervous symptoms are usually the most prominent. Headache is early and severe, vomiting often occurs at the onset and also later in the disease, and the patient rapidly passes into a state of stupor which is frequently disturbed by delirium or even mania. Convulsions may occur, and there is commonly incontinence of urine and faeces. The head becomes retracted, pressure in the suboccipital region produces severe pain, and Kernig's sign is almost invariably present. The pupils are usually dilated, and frequently sluggish in their reactions; ocular palsies are often an early symptom, but are less frequent than in tuberculous meningitis; nystagmus and ptosis are rarer. Swallowing may be difficult, owing to either spasm or palsy of the pharynx. Optic neuritis is relatively rare. Gross organic lesions of the central nervous system, as hemiplegia, paraplegia, and aphasia, are infrequent. The reflexes vary; the tendon-jerks are generally exaggerated, but may be unequal and variable, and in the severer cases may disappear. Babinski's sign develops in only a small proportion of the cases. The tempera-

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ture is very variable; the rise may be absent or negligible, or there may be extreme oscillations at short intervals. The pulse-rate is, as a rule, slow in relation to the temperature, and, like it, is often variable. In the early stages vaso-motor instability is one of the most noteworthy symptoms. Pain in the larger joints is also common at the onset, and arthritis or synovitis may occur as a complication. Pulmonary complications, pericarditis, and endocarditis are rarer. Rashes occur in a certain proportion of cases, often before the fourth day of the disease, as erythematous, papular, macular, or blotchy eruptions, which may become petechial or purpuric. Haemorrhagic eruptions have been common in some epidemics, and give the disease one of its titles. Herpes is often seen later.

There are four main **clinical types** of the disease: (1) The *abortive* form, which subsides gradually after 24–48 hours of illness of a mild septicæmic character, in which meningeal symptoms never develop. (2) The *acute* form, in which there is a sudden onset with meningeal symptoms, severe headache, retraction of the head, and signs of cerebral irritation passing into coma. In this type early and efficient treatment gives the best results, but recurrences are common and demand further treatment. A chronic condition may supervene. (3) *Chronic* forms are complicated by the formation of adhesions which obstruct the ventricles, or lead to encysted collections of pus in the cerebral or spinal meninges. (4) The *fulminating* types may be fatal in 48 hours or less. They set in suddenly with acute septicæmic symptoms—headache, vomiting, collapse, and purpuric eruptions, sometimes with a noisy delirium. Death may occur before the meninges become affected, but after death meningococci can generally be found in the ventricles or at the base of the brain.

The cerebro-spinal fluid.—The examination of this is essential for diagnostic purposes. Its character may vary in different stages of the disease. During the premeningitic or septicæmic stage it may be clear, or cocci alone may be found in it, or it may at first be turbid and later become clear. When meningitis has developed, the turbidity may vary from a faint opalescence to thick pus. The majority of the cells are polymorphonuclear, except in chronic cases with hydrocephalus and in posterior basic meningitis, in which they are chiefly lymphocytes. Its albumin content is increased and its normal reducing power is lost.

Prognosis.—Since the early and energetic administration of serum, intravenously in the septicæmic stage and intrathecally when the meningeal symptoms are developed, became general, the prognosis has become more favourable, both as regards mortality and after-results. The outlook is bad in fulminating cases with large purpuric rashes, and in those of acute onset with early loss of consciousness. A low blood-pressure signifies a severe infection. The appearance of signs of local lesions, as hemiplegia, and of nystagmus, which generally indicates a hydrocephalus, is ominous.

Treatment.—The best results are obtained by giving the specific univalent serum when the type of the infecting organism can be ascertained, but as no time should be lost, a multivalent serum should at once be administered intrathecally. In the premeningitic stages and in fulminating and severe cases with hæmorrhagic eruptions, intravenous injections of 200 to 600 c.c. are advocated. When there is frank meningitis the intrathecal injections are indicated in doses of 20–30 c.c. of serum twice a day for three or four days. A slightly larger quantity of cerebro-spinal fluid should be withdrawn before running in the serum (*see LUMBAR PUNCTURE*). Vaccine treatment has been satisfactory in some of the more chronic cases. The use of hexamine and of soamin has been disappointing.

POSTERIOR BASIC MENINGITIS OF CHILDREN

In this condition the infective organism is a meningococcus similar bacteriologically to that of meningococcal meningitis. It is a chronic encysted meningitis that occurs in children, generally under six months of age. The disease is sporadic, and is usually more common in the first six months of the year. It differs from cerebro-spinal fever of adults in the following respects: It has a more chronic course; rashes are rarer; opisthotonos is more marked; loss of vision without objective changes in the retina or optic nerves is more common; and the joint lesions tend to be peri-arthritis rather than intra-arthritis. The early symptoms may be indefinite, but there are usually fever, tremulousness, and bulging of the fontanelles. The prognosis is less good than in adults; about 50 per cent. recover, but 35 per cent. of these have some mental defect, blindness, or hydrocephalus.

Treatment should be carried out on the same lines as in adults. Occasionally the lumbar puncture proves dry, and then ventri-

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cular tapping and the intraventricular injection of serum may be attempted.

4. TUBERCULOUS MENINGITIS

Etiology.—Tuberculous meningitis is very rarely primary. The bacillus usually infects the meninges by the blood-stream, the source often being a tuberculous gland (generally mediastinal, less frequently mesenteric), or a focus in a bone; the petrous bone is frequently the site of disease. Many cases of otorrhœa are tuberculous.

Pathology.—The meningitis is found especially at the base of the brain, in the interpeduncular space and along the course of the middle cerebral artery. If the vessels are washed free of brain matter by a gentle stream of water, the tubercles can be seen in large number attached to the minute twigs. The exudate is rarely copious, is serous and somewhat gelatinous, often streaked with pus along the course of the Sylvian fissure. Caseous nodules are not infrequently found in the cerebellum or pons.

Symptomatology.—The disease is most common in children, and rarely occurs under the age of 2 years, though it has been met with as early as 3 months. The onset is characteristically insidious. After a period of malaise, marked by a disinclination of the child to play or move about, there is complaint of headache, and perhaps vomiting occurs. A slight rise of temperature may be found. Gradually drowsiness comes on, and in the course of one to two weeks this deepens into coma. A very common symptom at this stage is the "meningeal cry," a loud high-pitched scream emitted at irregular intervals without apparent cause. Twitchings of the face or limbs, or actual convulsions, are not uncommon, and often a hemiplegia or monoplegia is found. The neck muscles may be stiff, but there is not the emphatic retraction characteristic of meningococcal meningitis. The abdomen is often drawn inwards and assumes a scaphoid shape. Respiration becomes irregular and may be grouped, or approximate to the Cheyne-Stokes type. Kernig's sign may be present, but is not marked. Paralysis of the ocular muscles are common, especially of the external rectus. Ptosis may be seen. The pupils gradually dilate, and fail to respond to light. The optic disc may be somewhat blurred and the veins rather full, but there is rarely any approach to a pronounced neuritis; choroidal tubercles are occasionally found. The course usually

ends in death; recovery can, however, take place.

Tuberculous meningitis sometimes occurs in adults, and may be the terminal event in phthisis. The onset is often indicated by peculiar hysteroid symptoms and an early delirium and mental confusion, and in this condition patients even resemble persons under the influence of alcohol. Headache and vomiting may be present, and definite signs of cerebral mischief. A transitory aphasia may be an early symptom. In those cases which I have seen, coma has gradually supervened after about a week's illness.

Diagnosis.—It is well known that tuberculous meningitis is sometimes mistaken for other conditions. It must be remembered that the disease often attacks children apparently healthy. Under the age of one year the probabilities are greatly against tuberculous meningitis and in favour of meningococcal meningitis, more rarely influenzal meningitis, possibly syphilis or a serous meningitis. The association of otorrhœa with tuberculous disease of the temporal bone is important. In adults strange mental symptoms of rather abrupt onset, followed by drowsiness and coma, may suggest the disease. The diagnosis is made with certainty by the examination of the cerebro-spinal fluid. This is clear under pressure, reduces Fehling's solutions, contains an excess of lymphocytes and few, if any, polynuclear cells. On standing for twenty-four hours it deposits a cobweb film in which the tubercle bacilli can often be found.

Prognosis.—The disease is generally fatal, though recovery has certainly occurred. I have seen one case recover in which tubercle bacilli were found several times.

Treatment.—No treatment is known to affect favourably the course of the disease, though repeated lumbar puncture and the withdrawal of large amounts of cerebro-spinal fluid is certainly useful in many cases. Careful nursing is essential to avoid bedsores, and simple nourishing food must be given in the largest quantities possible. It is also necessary to attempt to allay the distress of the patient, more perhaps for the sake of the relatives than of the child, who is generally comatose; large doses of bromide, combined perhaps with chloral, often succeed, and serve also to check the convulsions that frequently occur.

W. B. WARRINGTON.

MENINGOCELE

MENINGITIS SEROSA (see HYDROCEPHALUS).

MENINGO-ENCEPHALITIS (see ENCEPHALITIS).

MENINGOCELE.—A protrusion of one or more of the membranes of the brain through an interval between the bones of the skull. If the protrusion contains brain matter it is called an encephalocele; should there be a prolongation into it from one of the ventricles of the brain, it is called a hydrencephalocele. These protrusions are congenital. The most common site is the occipital region, where emergence takes place from between the two lower centres of ossification of the squamous portion of the occipital bone. The next most common site is the root of the nose. Much less frequently a meningocele occurs in some other part of the median suture or on the lateral aspect of the skull. Rarely they protrude from the base of the skull in the region of the basisphenoid, where they may be taken for *naso-pharyngeal growths*, or from the neighbourhood of the cribriform plate of the ethmoid, where they are mistaken for *nasal polypi*. Many large meningoceles are accompanied by other developmental defects, and the patient does not survive. Meningocele form rounded fluctuating swellings, which pulsate, and become tense when the infant cries. The pure meningoceles are translucent; if there is contained brain matter they are opaque.

A meningocele at the root of the nose has to be distinguished from a *dermoid cyst* in this situation; dermoid cysts do not pulsate, and the intracystic tension does not vary. Most protrusions from the skull are best left alone, but a small pure meningocele may be removed, and the membranes sutured, with a very good prospect of success. For spinal meningoceles, see SPINA BIFIDA.

C. A. PANNETT.

MENOPAUSE.—The termination of the sexual life in women is marked by a syndrome of symptoms and a course of anatomical changes known as the menopause. There is no more critical time in a woman's life, for, apart from the troublesome symptoms incidental to the menopause, it is often the period at which certain morbid conditions of mind and body first make their appearance. The patient always requires careful observation and consideration.

MENOPAUSE

Onset and duration.—The onset of the menopause usually occurs between the ages of 47 and 52, and may last from a few months to a few years. Less commonly the first symptoms may be noticed at 40, or even as late as 55, but an extension of sexual activity beyond 52 is rare. A premature menopause is infrequently met with below 40, but in these cases as a rule there are certain pathological conditions, such as uterine or ovarian hypoplasia, which are recognizable as the cause. After operative removal of both ovaries the symptoms manifest themselves within a few months.

Symptomatology.—The outstanding symptom is an alteration in the menstruation, which takes one of three forms. Most commonly the flow gradually becomes irregular in quantity and periodicity. The intervals tend to become increased, and the amount lost is variable. Some months there may be a scanty flow, to be followed at the next period by a "flooding." Less commonly the flow diminishes gradually and regularly until complete disappearance; whilst rarely there is an abrupt cessation, after which there is no further loss.

A distressing symptom, apparently due to a lack of control of the vaso-motor system over the peripheral arterioles, is hot flushing of the skin. A wave of heat and redness, beginning in the face and head, passes downwards over the whole surface of the body, and is repeated many times a day. Other nervous manifestations are neuralgic headaches and incomplete digestive function, shown by flatulent dyspepsia, sluggishness of the colon, insomnia, and lassitude. Further, psychic disturbances are liable to occur, such as depression, instability of temper, unreasonable irritability, and lack of power of concentration. Most women suffer a certain amount of depression and discomfort at this epoch, and in some cases the symptoms become exaggerated and demand treatment. One of the most frequent of these exaggerated symptoms is an excessive and irregular red loss. It is most important, in that the bleeding may be set up by carcinoma, which is so prone to develop at this age that a careful examination should be made. Depression, too, may pass on to melancholia, or other form of insanity, the menopause being the time when mental disease is liable to show itself.

The **anatomical changes** are characterized by a partial atrophy of the genital system. The labia majora lose their fat and shrink,

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while the nymphæ almost disappear. The vulval mucous membrane becomes thin, dry, and smooth, with pale-yellow and red blotches, especially around the urethral orifice. There is a diminution in the length and width of the vagina, and the elasticity of its walls is lost. The vaginal cervix all but disappears, and the uterine body shrinks to little more than a mass of fibrous tissue. The ovaries and tubes also take part in the general atrophy. At this period also there is a marked tendency to obesity and to atrophy of the breasts.

Treatment.—In mild cases none is required, but where there is excessive blood loss, or nervous symptoms are severe, it is necessary to attempt some remedy. Menorrhagia is difficult to check, and scarcely responds to ergot and hydrastis, but is sometimes improved by pituitary extract, either in tabloid form or as hypodermic injection ($\frac{1}{2}$ –1 c.c. six-hourly). Calcium lactate is often disappointing. For distressing nervous symptoms, as insomnia, headache, and irritability, the best drug is a bromide in large enough doses to secure sleep. Hot flushes of the skin are controlled most readily by the addition of 5 min. of tincture of digitalis to the bromide mixture, the action of the drug having a steady influence on the easily dilated arterioles. Treatment by extracts of ovary or corpus luteum are disappointing as a rule, but, *faute de mieux*, may be given a trial. A better method is the administration of a polyglandular extract. It is most important to see to the general health. Constipation and indigestion require correction, and the patient should, if possible, be protected from all worry, anxiety, and uncongenial surroundings.

A. W. BOURNE.

MENORRHAGIA AND METRORRHAGIA, CAUSES OF.—Every woman has an average standard of menstrual loss during an average number of days, and should either of those averages be exceeded the loss is spoken of as *menorrhagia*. All uterine bleeding not associated with menstruation, whether occurring before puberty, intermenstrually, or after the climacteric, is best considered as *metrorrhagia*. Special names are often applied to such bleedings at the various epochs, but they signify little.

It is convenient to consider uterine bleeding under certain age-periods, and, when discussing the cause of the bleeding, to indicate and group the cases according to the period of life at

which the bleeding occurs. Thus the grouping here adopted will be:

1. Hæmorrhagic loss at puberty.
2. Hæmorrhagic loss during menstrual life.
3. Postclimacteric bleeding.

1. HÆMORRHAGIC LOSS AT PUBERTY

Excessive loss at puberty, especially if proved to be of uterine origin, is almost invariably menstrual, and only very rarely does a fibroid, polyp, or a new growth occur to give rise to metrorrhagia.

At puberty the onset of menstruation is usually irregular; one loss being followed by an interval of a few months, or even a year, before another loss occurs, the intervals gradually becoming shorter until rhythmic periodicity is established. It is not unusual for one or more of these losses to be decidedly excessive, and to last for a week or two, especially in anæmic, ill-nourished, and overworked girls. Treatment directed towards the anæmia by the administration of preparations of iron and salts and improvement of the hygienic conditions is usually successful in quietly establishing regular periodicity. In a certain number of cases, however, the menstrual loss is so prolonged and excessive as to cause blanching and syncopal attacks; this loss, when regularly repeated, may lead to death, which has been recorded in a few cases. No systemic disease is discoverable. For such a case a simple curetting is of evanescent benefit, and should be followed by the application of a cauterizing agent, such as pure nitric acid, to the endometrium, care being taken that the vagina and vulva are protected by lint saturated in a strongly alkaline solution of bicarbonate of soda. Even after this treatment failures may occur, in which case it is necessary to ligature the uterine arteries or even to remove the uterus.

2. HÆMORRHAGIC LOSS DURING MENSTRUAL LIFE

Bleeding during menstrual life may be either menorrhagic or metrorrhagic, depending often upon accidental factors as to type, even when the same cause is acting. It may be due to (i) general conditions, or (ii) local causes.

(i) The **general systemic conditions** nearly all produce a menorrhagia and comprise some anæmias (especially the pernicious form), scurvy, purpura hæmorrhagica, and, occasionally, uncompensated heart lesions. Hæmo-

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philia is usually included, but it is doubtful if the evidence at present available is sufficient to prove that hæmophilia exists as a disease of the female. Some psychical disturbances, chronic alcoholism, and the onset of Graves's disease are real causes. Lastly, altered climatic conditions, such as removal to a hot climate, often cause menstrual loss in excess, until accommodation takes place.

(ii) The **local causes** may, for convenience, and because of certain characteristic symptoms, be arranged under four headings:

- (a) New growths.
- (b) Displacements.
- (c) Inflammatory states.
- (d) Causes which relate to gestation

(a) Of **new growths**, the most important is *carcinoma of the cervix*, which usually causes irregular bleeding, accompanied by a characteristic discharge, in patients 30–45 years old, and more rarely at an advanced age. The diagnosis is at once established by palpation of the friable cervix, for which only an ulcerating polyp can possibly be mistaken: the loss is usually intermenstrual, but in the later stages the hæmorrhage is continuous.

Fibroids, fibroid polypi, adenomyomata, endometrial polypi, carcinoma of the corpus uteri, and chorion-epithelioma form a group which may be clinically indistinguishable among themselves; they all begin, in the first instance, as a menorrhagia, and subsequently the bleeding may be continuous. In the case of the myomata, the hæmorrhage, which is usually a prolongation of menstrual loss, depends upon the position and size of the growth. Thus, with a submucous fibroid of moderate size the hæmorrhage may be such as to cause blanching, while a subperitoneal fibroid occupying the whole abdomen will have no appreciable effect on menstruation. Again, the polypi, in relation to their size, produce in certain cases the more abundant bleeding, which is irregular and complicated by necrotic ulceration and foul discharge. Carcinoma of the body of the uterus is more commonly seen as a postclimacteric hæmorrhage, and chorion-epithelioma arises usually in relation to a recent pregnancy or puerperium, and is characterized by severe and irregular hæmorrhages. New growths of the adnexa, broad-ligament cysts, and pelvic tumours have no effect on menstrual loss.

(b) Among **displacements** a typical excessive period, prolonged for a day or two beyond the normal, is seen with *retroflexion* and *retro-*

version, and appears to be due to congestion which results from impeded uterine circulation. The loss is trivial in amount, and a diagnosis is readily made on examination. A more severe menorrhagia of recent origin is seen in cases of *subinvolution* of the uterus; in this condition a slight metritis is present in a uterus which, owing to lack of tone, becomes displaced dorsally. Menorrhagia from this cause is to be distinguished from the single brisk hæmorrhage encountered occasionally a month or more after parturition and due to the detachment of a small placental polyp.

(c) **Inflammatory conditions**, with the exception of fibrosis uteri, seldom produce marked loss. *Acute infections of the uterus and appendages* cause excessive menstrual loss, repeated at the next period. The chronic form of metritis—known as *fibrosis of the uterus*—produces abundant and prolonged menstrual loss, and ranks with a submucous fibroid, a polyp, and a malignant growth in its ability to produce a marked and dangerous secondary anæmia. For its relief nothing short of hysterectomy is to be relied upon.

Endometritis, *per se*, is no longer recognized as a source of hæmorrhage, although an acute endometritis is sometimes associated. In certain cases of fibroids and fibrosis of the uterus a much thickened, soft-felted, almost villous endometrium is encountered, and is of course the actual source of the bleeding, but is itself a manifestation of deeper-seated disease. An *erosion of the cervix* is an occasional cause of trivial and intermittent bloodstained discharge.

(d) **Bleeding related to gestation** occurs with a ruptured extra-uterine pregnancy, and is characterized by a variable period of amenorrhœa with subsequent sudden pelvic pain and bleeding in which the hæmorrhagic discharge soon becomes darker in colour. Bleeding in the early stages of pregnancy, when not due to a threatened abortion, is very rare. Carcinoma of the cervix and an erosion are occasionally found.

Climacteric bleeding.—Much that is incapable of substantiation has been said concerning bleeding at the climacteric. In the absence of any of the factors mentioned as responsible for bleeding during menstrual life, it may safely be assumed that as the ovaries atrophy, so the periods will intermit and disappear.

In the presence of excessive loss or bleeding it is advisable to find out the pathological condition responsible and treat accordingly.

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3. POSTCLIMACTERIC BLEEDING

Postclimacteric hæmorrhage is that which begins after a distinct menopause, and the later in life the hæmorrhage occurs, the more likely is it to be due (a) to malignant new growths, of which that affecting the body is now the commoner, and is the only malignant growth occurring in the nulliparous uterus, or (b) to polyp of endometrial or, less commonly, of fibroid origin. The symptoms are clinically indistinguishable from malignant new growths, and it may often happen that the curette used for diagnostic purposes fails to bring away typical specimens of either, leaving the diagnosis still in doubt. Hence many proceed forthwith to a hysterectomy in postclimacteric bleeding when carcinoma of the cervix has been ruled out. Incidentally, it is well to remember that bleeding resulting from a urethral caruncle, so common after the climacteric, has been frequently treated as of uterine origin.

Malignant transformation of a fibroid may be another source of bleeding, and is sometimes associated with pyometra.

BRYDEN GLENDINING.

MENSTRUATION, DISORDERS OF (see AMENORRHŒA; DYSMENORRHŒA; MENORRHAGIA AND METRRORRHAGIA).

MENTAL DEFICIENCY ACT, 1913.—

Mentally defective persons within the meaning of this Act are divided into four groups and defined as follows. *Idiots* are persons so deeply defective from birth or from an early age as to be unable to guard themselves against common physical dangers. *Imbeciles* are persons in whom there exists from birth or from an early age mental defectiveness not amounting to idiocy, yet so pronounced that they are incapable of managing themselves or their affairs, or, in the case of children, of being taught to do so. *Feeble-minded persons* are those in whom there exists from birth or from early age mental defectiveness not amounting to imbecility, yet so pronounced that they require care, supervision, and control for their own protection or for the protection of others, or, in the case of children, that they by reason of such defectiveness appear to be permanently incapable of receiving proper benefit from instruction in ordinary schools. *Moral imbeciles* are persons who from an early age display some permanent mental defect coupled with strong vicious or criminal propensities

on which punishment has had little or no deterrent effect. (For the symptoms and treatment of cases falling into these groups, see the article IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, MORAL IMBECILITY.)

How defectives may be dealt with.—

Persons defined as above may be dealt with under this Act by being sent to an institution for defectives, or by being placed under guardianship. This may be done at the instance of the patient's parent or guardian if he is an idiot or an imbecile, or at the instance of his parent if, though not an idiot or an imbecile, he is under the age of 21. In these circumstances two certificates have to be signed by two qualified practitioners, one of whom must be a medical practitioner approved for the purpose by the local authority or by the Board of Control, and also signed, when the defective is not an idiot or an imbecile, by a judicial authority recognized for the purposes of the Act, stating that the signatories of the certificates are severally satisfied that the person is a defective, and specifying the class to which he belongs, accompanied by a statement, signed by the parent or guardian, giving prescribed particulars with respect to him.

There are other circumstances in which a defective person may be placed under care:

(1) If he is found abandoned, without visible means of support, neglected, or cruelly treated.
(2) If he is found guilty of any criminal offence, or is ordered, or found liable to be ordered, to be sent to a certified industrial school. In this event the court may postpone sentence of committal to an industrial school and direct that a petition be presented to a judicial authority under this Act with a view to obtaining an order that he be sent to an institution or placed under guardianship; or, in lieu of passing sentence, the court may itself make an order which shall have the like effect.
(3) If he is undergoing imprisonment or penal servitude, or is in a place of detention by order of a court, or in a reformatory or industrial school, or in an inebriate reformatory, or is detained in an institution for lunatics or a criminal lunatic asylum; in any of these circumstances the Secretary of State, acting upon the certificates of two duly qualified medical practitioners, may order the transfer of the patient to an institution for defectives, or that he be placed under guardianship.
(4) If he is an habitual drunkard within the meaning of the Inebriates Act.
(5) If the local education authority notifies to the local authority

under the Act that an individual over 7 years of age is incapable of receiving benefit in special schools or classes, or cannot be instructed without detriment to the interests of the other children, or in respect of whom the Board of Education certify that there are special circumstances which render it desirable that he should be dealt with under the Act by way of supervision or guardianship; or if he is about to be discharged from a special school, on or before attaining the age of 16, and the local education authority is of opinion that it would be to his benefit that he should be sent to an institution or placed under guardianship. (6) If, being a woman, she is in receipt of poor relief at the time of giving birth to an illegitimate child, or when pregnant of such child.

Effect and duration of orders.—An order authorizes the conveyance of a defective person to, and his reception in, the institution named in the order, at any time within fourteen days (or, if the person is in a place of safety, within twenty-one days) after the date of the order. An order expires at the end of one year, unless continued by the Board for successive periods of five years upon consideration of reports and certificates submitted to it. If the defective person was sent to an institution or placed under guardianship before he was 21, the case is reconsidered by the visitors appointed under the Act, within three months after he attains that age. In cases where a defective has been placed by his parent or guardian in an institution, or under guardianship, it is lawful for the parent or guardian to withdraw the patient at any time on giving notice in writing to the Board, unless the Board determines within fourteen days after receiving the notice that further detention is required in the interests of the defective. If the Board does so determine, no further notice by the parent or guardian is allowed till after the expiration of six months from the last previous notice. The managers of any certified institution or house or approved home may discharge any defective placed there on giving one month's notice to the Board and to the parent or guardian of the defective. When the mental condition of a person detained in an institution for defectives becomes such, or is found to be such, that he ought to be transferred to an institution for lunatics, the Board, or the managers of the institution for defectives with the consent of the Board, shall cause steps for having a reception order, under the Lunacy Acts, made for his removal, provided that,

if the defective has been placed in the institution for defectives by his parent or guardian, opportunities have been given to the parent or guardian of taking such steps himself. On the other hand, when the mental condition of the patient in an institution for lunatics is found to be such that he ought to be transferred to an institution for defectives, measures may be taken to that end either by the Board or with its consent.

Any county court judge, police or stipendiary magistrate, or specially appointed justice who is a judicial authority for the purpose of the Lunacy Acts, is a judicial authority for the purposes of this Act.

The **central authority** constituted by the Act is the old Board of Commissioners in Lunacy, somewhat enlarged, and is charged with the general superintendence of matters relating to the supervision, protection, and control of defectives. The **local authority** for the purposes of the Act is the Council of the County or the Council of the Borough. The duties of a local education department are to ascertain what children within its area are defective; what children by reason of mental defect are incapable of receiving benefit from instruction in special schools or classes; and to notify the local authority of the names and addresses of defective children.

A person desirous of receiving defectives at his house for private profit may apply to the Board for a certificate, and the Board, if satisfied, may, on payment of a fee, grant a certificate subject to such conditions as they may impose: and a certificate so granted continues in force for the period for which it is granted, or until revoked or resigned.

E. D. MACNAMARA.

MENTAL TORTICOLLIS (see TORTICOLLIS AND WRYNECK).

MERALGIA PARÆSTHETICA.—Neuritis of the external cutaneous nerve of the thigh, named meralgia paræsthetica by Roth, is practically limited to the male sex. Pain referred along the front and outside of the thigh, aggravated by walking and standing, is often combined with soreness of the skin to touch, and at the same time diminished recognition of light pin-prick and finer tactile discrimination. The external cutaneous nerve pierces the fascia lata by means of a small tunnel, in which the nerve may become compressed or nipped,

MESENTERIC VESSELS, EMBOLISM AND THROMBOSIS OF

especially at its upper margin. This damage of the nerve may occur suddenly as the result of a muscular effort, as in a patient, a man of 63, who strained his right leg in pushing a heavy iron safe with his knee. Two days later, pain and soreness appeared along the front and outside of the thigh owing to a traumatic neuritis of the external cutaneous nerve. Sometimes the symptoms affect also the territory of the anterior crural nerve. They may be associated with alcoholism or diabetes, or other cause of toxic neuritis. **Treatment** by galvanism and ionization should always receive a good trial before resorting to the more drastic remedy of operating to free the nerve by incising the fascia lata at the point where it is pierced by the external cutaneous nerve.

WILFRED HARRIS.

MERCURY, POISONING BY (see POISONS AND POISONING).

MERYCISM (see STOMACH, FUNCTIONAL DISORDERS OF).

MESAORTITIS (see AORTITIS, SYPHILITIC).

MESENTERIC VESSELS, EMBOLISM AND THROMBOSIS OF.—The clinical manifestations produced by occlusion of the mesenteric vessels are rarely sufficiently distinctive to permit of unequivocal diagnosis. Only a certain proportion of vascular occlusions is followed by infarction, and even where infarction occurs it is rare for all the typical symptoms enumerated below to be present. Much less is it possible in the present state of knowledge to distinguish clinically between lesions arising from occluded arteries and occluded veins. Arterial occlusions appear to be rather more common than venous (when the survey is not limited to surgical cases), and in some instances simultaneous occlusion both of arteries and of veins occurs. Obstruction of the mesenteric veins is to be attributed to thrombosis; arterial obstruction may be due either to thrombosis or to embolism, and the latter is indeed the more frequent. While the slighter degrees of occlusion may lead to extreme engorgement, proceeding in some cases to limited ulceration or perforation of the intestine, it is only when complete stagnation of blood occurs that a gross necrosis (infarction) of a portion of intestine results. Hemorrhagic infarction of the intestine is the rule, anæmic infarction the exception. The superior mesenteric vessels are involved more frequently than

the inferior. Numerous cases were encountered in the late War as a result of gunshot injuries to the mesentery. Mesenteric occlusion is of rare occurrence in children.

Symptomatology and diagnosis.—The onset is sudden, with acute abdominal pain. Vomiting of bloodstained fluid and the passage by the bowel of a profuse quantity of blood are not infrequent. The patient may show signs of profound collapse. In other cases there is constipation, and the clinical picture is that of acute intestinal obstruction with the early addition of signs of peritonitis. Abdominal exploration reveals intense congestion or even commencing gangrene of the affected bowel. The presence of certain associated lesions may be of material assistance in arriving at a diagnosis. Of these the more important are valvular lesions of the heart (arterial embolism or venous thrombosis), degenerative diseases of blood-vessels (arterial thrombosis), cirrhosis of liver (downward spread of thrombosis from portal vein), appendicitis, and inflammation of the female pelvic organs (infective thrombosis of veins). Evidence of preceding or simultaneous embolic occlusion of other vessels would further strengthen the probability.

Treatment.—Laparotomy, unless some such condition as ulcerative endocarditis forbid, should be performed as soon as possible. Resection of the affected portion of the intestine and mesentery, when this is possible, holds out the only hope of recovery. In cases secondary to an infective focus, such as an inflamed appendix, the infective focus should also be removed.

C. E. LAKIN.

METHÆMOGLOBINÆMIA (see CYANOSIS).

METHÆMOGLOBINURIA (see URINE, EXAMINATION OF).

METRITIS (see ENDOMETRITIS, ENDOCERVITIS AND METRITIS).

METRORRHAGIA (see MENORRHAGIA AND METRORRHAGIA).

MICROCEPHALY (see SKULL, CONGENITAL ABNORMALITIES IN SHAPE OF).

MICROPHTHALMOS (see EYE, CONGENITAL ANOMALIES OF).

MICROSPOROSIS (see RINGWORM).

MICTURITION, FREQUENCY OF (see URINE, VARIATIONS IN AMOUNT OF).

MIDDLE-EAR DISEASE (see OTITIS).

MIGRAINE.—Hemicrania, or migraine, or megrim, is a vaso-motor neurosis of the pia arachnoid and cortex. Heredity is extremely common in this disease, and in certain families migraine often alternates with other neuroses such as epilepsy, insanity, hysteria, and somnambulism. It is often said to be especially common in highly intellectual subjects, but analysis of private and hospital records scarcely bears this out. As a rule, migraine can be traced back to the early years of childhood, when the attacks are apt to be known as bilious headaches.

A characteristic attack of migraine consists of unilateral headache spreading from the eyebrow and forehead over the top and back of the head.

Symptomatology.—Often beginning as a dull pain on awakening, the headache increases in intensity to such a degree that the sufferer may have to lie down in a darkened room with all noise and vibration excluded as much as possible. The pain may last for twelve to forty-eight hours, when vomiting follows. In some cases vomiting occurs early in the attack and may continue for hours, during which the pain continues unabated. For migraine that is associated with indigestion, see STOMACH, FUNCTIONAL DISORDERS OF.

The more typical cases of migraine have various warnings before the onset of the headache. A few notice, some hours before the onset of the attack, an excessive sense of well-being, and perhaps inordinate appetite. A fairly common precursor of the headache is a visual scotoma, which may take various forms. In some cases the sudden onset of homonymous hemianopia ushers in the attack, vision recovering after about twenty minutes, and then the headache commences on the opposite side to the blind fields. In others, blurring of central vision begins the attack, the scotoma opening out in horseshoe form towards the periphery of the visual field, with prismatic colours disposed in angular formation in its margins, the so-called "fortification spectrum," or scintillating scotoma. Either unilateral or general headache usually follows after twenty minutes or half an hour. Occasionally a psychical aura is met with, or an intellectual aura in which a certain scene recurs in each attack, apparently before the eyes of the sufferer. Bilateral hemianopia causing temporary complete blindness is very rarely described as a

warning. In a few subjects, tingling is felt along the lips and tongue, with aphasia when the symptoms are right-sided; and in a small percentage of cases, paresis of an arm, and rarely of a leg, may also be experienced for a quarter of an hour or so. Occasionally such warnings occur without the subsequent headaches; I have known an instance of a man who suffered severely with migrainous headaches preceded by a hemianopic scotoma, whose son never had the headache but on three occasions experienced complete hemianopic blindness of a temporary character.

The **pathology** of the disease is speculative, but in all probability the stage of the aura, or warning, consists of a vaso-motor spasm affecting the meninges and cortical vessels, especially in the occipital area. In this stage ophthalmoscopic observation of the retina may show narrowing of the arteria centralis retinæ, succeeded, when the headache sets in, by dilatation of the vessel, with throbbing of the carotid and temporal arteries. This headache, which is agonizing and excruciating torture to some patients, is possibly due to dilatation of the cortical and meningeal vessels succeeding the previous spasm. On two occasions I have known the hemianopic aura of migraine become permanent blindness, which is suggestive of vaso-motor spasm producing thrombosis in the half-vision centre. A curious symptom experienced by a few sufferers from migraine is a hemianopic dream in which there occurs at regular intervals the same scene, a house or landscape, of which only one half is visible. In some other sufferers from migraine, terrifying dreams, usually of fire, may precede an attack.

There is undoubtedly a close connexion in many cases between migraine and epilepsy; the two diseases may alternate in the same family, and indeed in the same patient. Moreover, the visual aura of migraine—namely, the scintillating scotoma or hemianopia—may precede the fits as well as the ordinary migrainous attacks in these patients. The headache, too, which usually follows epileptic fits recalls that of migraine. Finally, migraine, like epilepsy, is often brought on by excitement, worry, hot stuffy rooms, or heavy meals.

Quite an appreciable proportion, perhaps 10 per cent., of sufferers from migraine have it in a form which I term *migrainous neuralgia*. In these cases the painful attacks may be practically limited to one side of the forehead, temple, and cheek; it is not uncommon for

MIKULICZ'S DISEASE

this condition to be mistaken for chronic trigeminal neuralgia. However, questioning will usually elicit the fact that 5-10 per cent. of the total number of attacks occur on the other side, though always with much less severity; while trigeminal neuralgia is only rarely bilateral. The pain is more constant than that of trigeminal neuralgia, lasting for hours, and not brought on by eating, talking, or other movements of the face or jaw. Sufferers from this form of neuralgia will usually confess to a sensation of nausea during the attack, even if actual vomiting does not occur. Tenderness over the eyebrow, temple, or anterior scalp is the rule.

Treatment.—Rest in bed, with the room darkened and with perfect quiet, is always best for a bad attack of migraine. Food, as a rule, is not tolerated during the pain, but occasionally considerable relief is brought about by a dose of aspirin or amidopyrin; in severe cases 3 or 4 gr. of veronal and $\frac{1}{10}$ gr. of heroin may be added. Preventive treatment often succeeds in diminishing the frequency or severity of the attacks. Bromide is the most valuable drug for this purpose, and in some cases considerable benefit follows a course of bromide with a little strychnine and nitroglycerin.

WILFRED HARRIS.

MIGRAINOUS NEURALGIA (see NEURALGIA).

MIGRAINOUS OPHTHALMOPLÉGIA (see OPHTHALMOPLÉGIA).

MIKULICZ'S DISEASE.—Chronic bilateral swelling of the lachrymal and salivary glands, of unknown origin.

Since the first description by Mikulicz the term has been used to include other conditions in which a bilateral swelling of these glands forms only a part of the picture, and the following forms have been indicated: (1) Symmetrical swelling of the salivary and lachrymal glands; (2) symmetrical swelling of the salivary glands alone; (3) symmetrical swelling of the lachrymal glands alone; (4) symmetrical swelling of the lachrymal and salivary glands, or of both, in association with enlargement of the spleen or lymphatic glands; (5) similar swelling of the lachrymal and salivary glands with alterations in the blood, indicative either of a severe *anæmia* with lymphatic pseudo-leukæmia and aplasia of bone-marrow, or of true leukæmia.

Etiology.—The association of inflammatory

conditions of the mouth, nose, and pharynx suggests an infective origin for true Mikulicz's disease, whether the symmetrical enlargement of salivary and lachrymal glands occur alone or is accompanied by enlargement of the lymphatic glands. Rarely, the syndrome appears to be congenital, hereditary or familial. Gout, tubercle, syphilis, and, as has been mentioned, leukæmia, are other causes. Sialodochitis fibrinosa is an unusual affection which may produce part of the syndrome; there is symmetrical enlargement of one or more pairs of salivary glands, generally of the parotids, but the swelling occurs intermittently, is sudden in onset, often provoked by the taking of food, and sometimes painful at first and accompanied by fever. It is due to a fibrinous inflammation of the salivary ducts, and plugs of fibrin have been removed from the orifices of the ducts, from which they projected into the mouth.

Prognosis.—In true cases disfigurement is the only important disability. This and the slight occasional discomfort disappear in the course of a few years. The prognosis of cases in which the syndrome is symptomatic of leukæmia, gout, syphilis, or tubercle is essentially that of the primary disease.

Treatment.—Any local source of chronic infection, such as oral or nasal sepsis, should be attended to, and the general health should be improved as much as possible. Arsenic in large doses appears to be the most successful remedy. Exposure of the swellings to X-rays leads often to improvement and sometimes to cure. Eradication by surgical means has been employed. For the syphilitic and tuberculous cases appropriate treatment is, of course, indicated. Potassium iodide has been found beneficial, even in the absence of ascertained syphilis.

FREDERICK LANGMEAD.

MILITARY TUBERCULOSIS (see TUBERCULOSIS, ACUTE GENERAL).

MILITARY TUBERCULOSIS, ACUTE (see PULMONARY TUBERCULOSIS).

MILITARY TUBERCULOSIS OF PERITONEUM, ACUTE (see PERITONITIS, TUBERCULOUS).

MILROY'S DISEASE (see EDEMA).

MINERAL WATERS (see HYDROTHERAPY; SPA TREATMENT).

MINER'S LUNG (see PNEUMONOCOINOSIS).

MINER'S NYSTAGMUS.—An affection of the nervous system in which the eye symptoms may be more pronounced than those of other parts. It is found among coal miners, and, although many observers say that it occurs in no other occupation, I believe that well-authenticated, though isolated, cases have been reported among ironstone miners and plate-layers. Since 1907 it has been scheduled as an occupational disease under the Workmen's Compensation Act.

Etiology.—The three most important factors are (1) defective illumination due to the low candle power of the safety lamp, (2) refractive errors of the eye, and (3) the frequent injuries which miners sustain during their work, more especially injuries to the eye itself.

Heredity appears in some cases to be a predisposing cause.

Symptomatology.—Miner's nystagmus should be regarded not as a disease limited to the eyes, but as a general nervous disease of which one symptom is nystagmus or oscillation of the eyeballs. In a typical case other symptoms are generally found, namely, headaches, nausea, attacks of giddiness, sleeplessness, and general neurasthenic symptoms such as nervousness, arterial throbbing, a quickened pulse, exaggerated knee-jerks, and increased vaso-motor irritability. Mental symptoms of a serious nature occasionally occur. The subjective eye symptoms are failure of sight, photophobia, and night-blindness, but the most characteristic complaint is that the lights in the pit, or on the surface at night, jump about or flicker.

Physical signs.—The most important physical signs, besides the nystagmus, are clonic spasm of the eyelids, muscular tremors, especially of the head and eyebrows, and a characteristic gait; the patient walks stiffly, endeavouring to compensate for his visual defect by throwing the head backwards, keeping the eyes down with drooping eyelids. The movement of the eyeballs is rotatory, and involuntary. Both eyes are nearly always affected, but the movement may be different in direction. The oscillations vary from 100 to 300 per minute, and in the vast majority of cases both eyes move at the same rate. Clonic spasm of the eyelids is a common physical sign, and sometimes the most prominent. It may be an early sign, but is sometimes a late one, and often it remains after the nystagmus has ceased.

Diminution of visual acuity and contraction of the visual fields, especially for white, blue,

and red, are usually present. Paralysis of accommodation exists in nearly all typical cases, and injection of the eyelids and the eyeballs is frequently found.

Diagnosis.—It is most important to decide whether the symptoms are due to the patient's employment or not. Oscillation of the eyeballs is associated with several diseases of the brain and spinal cord, notably Friedreich's ataxia, disseminated sclerosis, and cerebellar lesions, as well as with some congenital diseases of the eye, but the history of the case and the presence of other symptoms should be sufficient to make the diagnosis of miner's nystagmus certain. In no other condition are oscillation of the eyeballs and clonic spasm of the eyelids associated.

Treatment.—In severe cases the patient should give up work completely, have a prolonged rest, and never return to work under ground. In all cases refractive errors should be corrected by glasses, and the general neurasthenic symptoms treated on the usual lines. Prevention consists in increasing the illuminating power of the safety lamp, improving the ventilation of mines, and excluding from them men with refractive errors.

FRANK SHUFFLEBOTHAM.

MIOSIS (*see* EYE, EXAMINATION OF).

MISCARRIAGE (*see* ABORTION AND MISCARRIAGE).

MITRAL INCOMPETENCE (*see* VALVULAR DISEASE, CHRONIC).

MITRAL STENOSIS (*see* VALVULAR DISEASE, CHRONIC).

MOIST GANGRENE (*see* GANGRENE OF THE EXTREMITIES).

MOLAR PREGNANCY (*see* PREGNANCY, MOLAR).

MOLES.—A mole is a congenital localized alteration of the colour or structure of the skin, generally with increased pigment and abnormal growth of hair. (In the Continental sense of the term, all congenital circumscribed growths of the skin are *naevi*.)

Etiology and pathology.—Moles are due to some developmental error of growth, and are probably always present at birth, although they may not become visible until some weeks or months or even years afterwards. They are often attributed by the mother to maternal impression. Histologically, "soft" moles are

MOLES

characterized by collections of large oval cells situated in the corium which have become separated from the surface epithelium. When malignant degeneration occurs in a mole the resulting growth is a carcinoma. "Hard" or papillary moles come into a different category; they form papillomatous outgrowths which, microscopically, show thickening of the horny layer and epidermis, and are often arranged in unilateral lines or bands.

Symptomatology.—Moles vary enormously in character according to the particular skin structure predominantly involved; they are also very variable in number, in size, and in shape. A common form which occurs on the face or back or other part of the body is a smooth pigmented lenticular patch often covered with fine or coarse hairs (*navus pilosus*). This variety of mole may affect large areas of the body such as the "bathing-drawers area," causing great disfigurement. Another type is raised and fleshy owing to the development of fatty and connective tissue (*navus lipomatodes*), and the surface may be ridged or furrowed like the convolutions of the brain (*cerebriform navus*), or mamillated or warty (*navus papillomatousus*, *navus verrucosus*, etc.).

The hard papillary mole also varies in appearance and extent. Often it takes the form of lines or bands made up of wart-like projections (*navus linearis*, etc.); these may be soft and of the same colour as the skin, or horny and deeply pigmented. They are usually unilateral, but may occur on both sides of the body. Probably many of the cases described as ichthyosis hystrix, in which there are large pigmented horny projecting masses, may be regarded as linear naevi.

Treatment.—The treatment of moles depends upon their size and number and situation. Small pigmented moles may be removed with the electrolytic needle, using the negative pole and a current of 1-3 ma. If there are coarse hairs, these may be removed in the same way, and the rest of the mole will usually disappear. For larger non-hairy moles the application of solid carbon-dioxide snow is a good method. A stick of the same diameter as the mole is applied to it with firm pressure for about thirty seconds, and the application is repeated at intervals of about three weeks until the lesion has disappeared. Since the application is followed by a rather severe reaction, large moles can only be dealt with piecemeal, and many applications may be required. Alternative methods are painting

MOLLUSCUM CONTAGIOSUM

with chemical caustics, such as nitric or trichloroacetic acid, excision followed by Thiersch grafts in the case of moderately large moles, and treatment by means of the galvano-cautery, diathermy, radium, or X-rays. Very extensive cases are best left alone, and it should be remembered that any method which falls short of complete destruction of the growth, especially in the deeply pigmented moles of adults, may lead to malignant changes. Any mole in an adult that shows signs of active growth should be freely excised without delay.

S. E. DORE.

MOLLITIES OSSIUM (see OSTEOMALACIA).

MOLLUSCUM CONTAGIOSUM.—An infection of the skin causing the formation in it of small tumours varying in size from a tiny dot to a pea, often arranged in groups. As the name indicates, they are contagious, as has been proved by experiment. The infective agent is probably ultramicroscopic. Children are more often affected than adults. The histological appearances are characteristic; the cells of the Malpighian layer proliferate, giving rise to an ovoid mass which raises up and flattens out the surrounding epithelium. The centre undergoes a peculiar degeneration, forming lobules containing a curdy mass of degenerated cells, the "molluscum bodies." Not infrequently an inflammatory reaction from coecal infection occurs; the original lesion is thereby considerably modified, an inflamed papule replacing the pearly umbilicated sessile tumour. In the groins and on the scalp, in rare instances, a hypertrophic form may be met with, resulting in large tumours bearing a superficial resemblance to a malignant growth. **Treatment** consists in evacuating the contents by simple pressure or after incision. If the latter plan be adopted, care should be exercised to avoid damaging the healthy skin and causing scarring. If the area is previously painted with tincture of iodine, and after the little operation a second application is given, the likelihood of cure is increased. Some prefer to use the curette or cautery. When the lesions are in the neighbourhood of the eye, precautions should be taken to avoid conjunctivitis. When secondary infection takes place, cure usually results. The giant forms demand free excision.

H. MACCORMAC.

MOLLUSCUM FIBROSUM (see SKIN, FIBROMATA OF).

MONOPLÉGIA.—This is a term applied to paralysis of one limb, and may depend on many different pathological conditions that involve either the upper or the lower motor neurones concerned in the innervation of the limb. It is, however, usually limited to spastic paresis due to disease of the upper motor neurones.

There may be either a crural or a brachial monoplegia; its most common cause is a vascular lesion that damages the corresponding motor area, since focal lesions of the pyramidal tracts below the cortex are less likely to pick out the fibres destined for the voluntary control of one limb only. In most cases the restriction to one limb is only apparent; when there is a brachial monoplegia the reflexes of the homolateral leg are usually disturbed, or careful examination may reveal a slight paresis.

A local lesion of the grey matter of the cervical cord can produce paralysis limited to one arm, but this is a flaccid and atrophic weakness. The most typical cases are those in which a lesion of one lateral column below the first dorsal segment causes paralysis of the homolateral leg, but even here the symptoms are not strictly limited, as sensory disturbances of the Brown-Séquard type can be usually found on the opposite side.

One of the most common causes of monoplegia is disseminated sclerosis, as a patch of the disease may for a time affect one limb only.

GORDON HOLMES.

MONSTERS.—A monster is defined as a child born with a deformity so pronounced as to interfere with the general or local development of the body.

Monsters may be single or double. Single ones are more common than double, and on the whole are more liable to give rise to difficult labour, as the double monster is usually born prematurely. Aborted ova show a comparatively high percentage of monsters.

Etiology.—Heredity plays a part in the production of monsters. Injury to the uterus or disease of the uterus or membranes may do so likewise. Development of the fœtus may be interfered with at an early stage by the circulation of abnormal chemical substances in the blood of the mother. Syphilis is responsible for some cases.

Diagnosis.—Before the onset of labour, abnormalities such as anencephalus may be suspected on abdominal examination. The presence of hydrannion or exceptional abdomi-

nal enlargement suggests the possibility of monster-formation, though the condition is usual in multiple pregnancy. In cases in which Cæsarean section is proposed as a means of delivering a patient with small pelvic measurements, if the foetal parts cannot be defined, or if the abdomen is unduly large, resort to an X-ray examination should be made before operative measures are undertaken. Double monsters are not diagnosable by palpation, except perhaps as twins.

During labour, unusual delay in the entrance to the pelvis of the presenting part, or, after a portion of the child has been born, secondary delay, leads to suspicion. In these circumstances internal examination under an anæsthetic should be thorough, the whole hand, if necessary, being introduced into the uterus. As abnormalities are frequently multiple, such an appearance as that of spina bifida in a breech case should prepare one for further abnormality.

Treatment.—Satisfactory treatment for the mother, in all cases of dystocia caused by the presence of a monster, depends on accurate diagnosis. As the child need not be considered, ample time should be given for spontaneous delivery, if this is likely to occur. If not, the reduction in size of the parts which will not pass through the maternal passages should be complete before delivery is attempted.

Varieties.—The most common forms of single monster are:

1. Those due to incomplete closure of the medullary canal, the most marked example being the *anencephalic* fœtus. In this the vault of the cranium is absent, the brain undeveloped, the neck rigid and the shoulders broad. The fœtus usually presents by the face, and delivery is often spontaneous. There may be difficulty in the passage of the shoulders, partly because they are unduly broad and partly because the ill-developed head has caused only partial dilatation of the cervix.

Cleidotomy, double if required, should be performed, and gentle traction applied to the head with the cranioclast, the cervix being eased over the shoulders with two fingers of the free hand. Delivery by the breech is simple in these cases, so that podalic version should be performed where practicable. A diagnosis is frequently not made early enough for this.

2. The *acardiac* monster, occurring essentially as a parasite in twin (uniovular) pregnancy. There are three kinds: (a) the *acephalic* (without head); (b) the *acornic* (with undeveloped head

and the rudiments of a body); (c) the *amorphic*, a shapeless mass without head or limbs. The only condition under which such monsters produce dystocia is when the mass becomes œdematous. They must then be removed piecemeal.

3. Those due to incomplete closure of the lateral body plates. Extrusion of certain viscera occurs, and in its complete form there is no abdominal wall and the viscera project into the amniotic cavity.

Difficulty in delivery is sometimes experienced because the cord in these cases may only be 2-3 in. in length. The defect can be diagnosed only by palpation internally. The cord should be severed and the fœtus delivered by the breech.

Double monsters are all derived from one ovum.

Infinite modifications of the double monster are possible. They are divided into three main classes:

1. Two separate children joined at some part of the trunk. *Thoracopagus* and *ischiopagus*, the former joined in part at the thorax, the latter at the sacrum, are the best known. The Siamese Twins come under the heading of *thoracopagus*.

2. Two heads joined to one body—*dicephalus*.

3. Two separate bodies joined at the head—*syncephalus*.

Delivery of double monsters is sometimes spontaneous, as premature delivery nearly always occurs.

The monsters of groups 1 and 2 usually present by the breech, and delivery is more simple when this is so. If either condition is suspected, the posterior head should be encouraged to enter the pelvis first, by throwing the body or bodies forward over the mother's abdomen. If the heads present, one is born first, and spontaneous evolution is said to occur round the neck of the other. Impaction is more likely to occur, when it is best to amputate the first head and perform version.

Unless a syncephalic monster is considerably premature, delay is sure to be caused by the head, whether this presents or comes last. It is to be treated by the same measures as any other abnormally large head, i.e. by perforation and extraction.

FRANCES M. HUXLEY.

MORAL IMBECILITY (see IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, MORAL IMBECILITY).

MORBILLI (see MEASLES).

MORBUS CÆRULEUS (see HEART, CON-
GENITAL DISEASE OF).

MORBUS CÆLIACUS (*syn.* Celiac Disease, Acholia, Celiac Infantilism, Intestinal Infantilism).—A disease of obscure origin occurring in children, and characterized by chronic or relapsing fatty diarrhoea, enlargement of the abdomen, and retardation of physical growth.

Etiology and pathology.—The obscure origin of the disease must at present be emphasized; the same condition produced by abdominal tuberculosis is not celiac disease. At autopsies, which are rare, it is the rule to find nothing in the liver, pancreas, or intestines which accounts for the disease. The variety of the superabundant fat in the stools, which is partly neutral fat but mainly split fat, suggests that the function of the bile is more at fault than that of the pancreatic secretion. Possibly there is a deficiency of bile-salts. Clinical tests for pancreatic disease are negative. The theory that the disease is due to enteritis from abnormal bacteria in the intestine is not supported by any constant evidence.

Boys and girls seem about equally affected. Most commonly the disease starts in the second or third year of life. It is possible that there are transient cases of the same type, but only the chronic cases will here be described.

Symptomatology.—The onset is usually insidious, but occasionally is abrupt, starting with vomiting and diarrhoea, of which the latter persists. The disease once established, the pallor of the stools is soon manifest.

Physical growth stops during the acuter stages, and after several years the stunting in development is very noticeable. The child may be the height of one only half its age. The weight usually corresponds to the height rather than to the age of the child, but in the chronic stages the patient is not wasted. The abdomen is large, pendulous, and soft on palpation. Nothing abnormal can be felt within it.

The stools are typical—large, pale, offensive, unformed. The excess of fat in them accounts for their porridge-like consistency and colour. Undigested proteins and starch may be present from intestinal hurry. Notwithstanding the bowels may move only once or twice daily, there is excessive intestinal elimination, which is the cause of the retardation in growth.

MORBUS CÆLIACUS

Mucus in the stools is never very considerable ; there is no macroscopic blood. Under treatment the stools should become darker, more solid, or even formed.

The tongue is usually clean except in diarrhoeal exacerbations. Ulcers in the mouth may be very troublesome. The appetite is usually poor, often very capricious. Perverted tastes in food are common ; soil-eating (pica) may occur. Absolute refusal of food, simulating anorexia nervosa, may be found. The difficulty in feeding these children is frequently a great bar to their progress.

The temperature is often slightly raised for weeks together. If all else is going well, this should be disregarded.

Functional nervous symptoms are very common. There is exaggeration of all actual ailments and imagination of others. Weakness of the legs is a frequent complaint. The knee-jerks are often unobtainable ; this is due, I think, to muscular atony. I have known two cases diagnosed as infantile paralysis. Flat-foot may occur, or even late rickets. Tetany has been reported.

Diagnosis.—In the earliest stages diagnosis is difficult, but later the manifestation of the cardinal features makes it easy to anyone familiar with the condition. Abdominal tuberculosis and pancreatic disease must be excluded.

Prognosis.—The disease may last for many years, and though there is a gradual improvement in digestive capacity and development is re-established, only a minority of the patients appear to reach the normal height. Death from the disease itself is very rare ; from intercurrent diseases it is uncommon. Throughout there is very slow progress as a rule, and disheartening relapses occur.

Treatment.—Only the principles of treatment can be given here. Bed is indicated if there is steady loss of weight from protracted diarrhoea or defective intake of food.

Of first importance is the *diet*, in view of the defective fat-digestion. Fats—milk, butter, fat, suet—must be reduced or eliminated ; in addition, green vegetables and fruits are usually too laxative for these cases. The foods that may be given are meat (preferably minced raw and then cooked), chicken, fish, tongue, lean ham, eggs (one or two daily), potatoes, toast, biscuits, rusks, a little bread, honey, syrup, home-made potted meat, sweet jellies, egg jelly, sponge-cakes or sponge-puddings. For drinks, water, chicken-broth, bovril, skimmed

MOUNTAIN SICKNESS

milk. On improvement, very small quantities of butter, custard, milk puddings, and jellies may be added, and, after these, milk to drink. On some such diet, low in fats, these children must continue. It is, however, a mistake to persist too long with too strict a diet. The patients do better (i.e. grow faster) when passing a slight excess of fat in the stools than when this is prevented by prolonged dieting. During hot weather stricter measures are advisable.

In some recent cases I have been pleased with the effect of the administration of bile-salts (2 gr. t.d.s., p.c.), which has caused a considerable gain of weight with a diminution of fat in the stools.

Drugs may be of service. Bismuth carbonate, soda, and a trace of rhubarb form a useful stand-by. Opium may be necessary in acute stages. Castor oil, given occasionally, is very helpful. All irritant purges are contraindicated. Aperients are rarely needed, and a little fruit may serve this purpose, unless disturbed digestion is present.

Ulcers in the mouth may be treated by hydrogen peroxide as a mouth-wash and potassium chlorate internally.

No drugs seem to stimulate the appetite. A few days' rest in bed on a low liquid diet may help ; or in worse cases a complete change of environment. I have known forcible feeding to be required.

REGINALD MILLER.

MORBUS MACULOSUS (see MELÆNA NEONATORUM).

MORPHIA POISONING (see POISONS AND POISONING).

MORPHEA (see SCLERODERMIA).

MORVAN'S DISEASE (see SYRINGOMYELIA).

MOUNTAIN SICKNESS.—A disorder produced by lack of oxygen (anoxæmia) resulting from the low atmospheric pressure encountered in high altitudes. It is met with in aviators as well as in those who make mountain ascents.

Symptoms.—The usual symptoms are headache, dizziness, nausea, hyperpnoea, and cyanosis. There may be a sense of great prostration, accompanied by vomiting, palpitation, and ringing in the ears. The pulse is rapid, weak, and intermittent, and syncope may follow. Epistaxis is an occasional symptom. The temperature may be slightly raised. After a time the low atmospheric pressure is

MULTIPLE NEURITIS

compensated for by greater activity of the alveolar epithelium, by improved interchange in the alveolar air, and by increase in the red blood-corpuscles and hæmoglobin content of the blood. After acclimatization the symptoms disappear, but may return on exertion.

Treatment.—Rest until compensation is established is all that is necessary in most cases. Oxygen is beneficial, but seldom available.

FREDERICK LANGMEAD.

MOUTH-BREATHING (see DENTITION AND DENTAL IRREGULARITIES; NASOPHARYNGEAL OBSTRUCTION).

MOUTH, DISEASES OF (see STOMATITIS AND GLOSSITIS).

MOVABLE KIDNEY (see VISCEROPTOSIS).

MUCOUS COLITIS (see COLITIS).

MUCOUS POLYPI (see UTERUS, POLYPOI OF; NASAL POLYPI).

MULTIPLE IDIOPATHIC HÆMORRHAGIC SARCOMA OF KAPOSI (see SKIN, MALIGNANT NEW GROWTHS OF).

MULTIPLE NEURITIS.—Inflammatory changes involving the fibres of the peripheral nerves may arise from a great variety of causes. They may be due to exogenous poisons, to endogenous poisons arising through metabolic disorders, or to toxins engendered in the course of bacterial infection.

The *exogenous* poisons, in the order of frequency, are alcohol, lead, arsenic, and more rarely mercury, copper, carbon bisulphide, anilin, phosphorus, and certain ptomaines. *Endogenous* poisoning may occasion neuritis in gout, diabetes, in foods deficient in vitamins (causing beriberi), and many forms of gastrointestinal disturbance. Neurotoxins are occasionally formed in jaundice, in cirrhosis of the liver, and in atheromatous and senile degenerative conditions. Acute infections, as typhoid, scarlet fever, influenza, diphtheria, erysipelas, and rarely whooping-cough, may produce neurotoxins; and toxins are formed in wound infections, puerperal sepsis, putrid bronchitis, acute rheumatism, syphilis, and also, though seldom, in tuberculosis and in pneumonia. One form is probably due to a specific infection, but the organism has not been isolated.

Multiple neuritis sometimes occurs without any discoverable cause.

Symptomatology.—The symptoms may be most conveniently studied in connexion with

alcoholic neuritis, which will accordingly be considered first. It is essentially a disease of the habitual spirit-drinker, and appears to be determined by the ingestion of excessive quantities of alcohol over very long periods, since a short period of drinking sufficiently intense to lead to delirium tremens does not, as a rule, tend to produce it. The prodromal symptoms are formication and pain in the limbs, which become tender. Muscular weakness appears and rapidly becomes more pronounced, and in the course of a few days may lead to complete loss of power in the affected limbs. The legs are more frequently and more intensely affected than the arms. The muscles involved are flaccid, very tender, and soon begin to waste. The wasting is occasionally masked at first by the presence of œdema. The nerves are also tender and sometimes feel thickened.

All the deep reflexes, and not only those of the paralysed extremities, are, as a rule, absent. The paralysis does not usually involve all the muscles of a limb; its distribution corresponds generally with that of the peripheral nerves affected, but it is by no means rare for the fibres supplying one or more muscles to escape, and sometimes only a portion of a muscle is paralysed. Certain nerves suffer more frequently and intensely than others; thus, in the legs the peroneal and posterior tibial nerves are those most frequently affected, and in the upper extremity the posterior interosseous. Generally speaking, the nerves supplying the most distally situated muscle-groups are the most severely involved.

The degree of disturbance of sensation is very variable; the nerves of muscle and joint sensibility corresponding to the paralysed areas also suffer, and often their recovery does not take place until long after the restoration of voluntary movement is nearly perfect. Cutaneous sensation may be entirely lost in the area of the cutaneous nerves that correspond with the distribution of the muscular paralysis, but only slight diminution of sensibility may accompany very marked motor disturbance. Very often areas of hyperæsthesia exist, either contiguous to anæsthetic areas or apart from other sensory disturbance. The hyperæsthesia may be very intense, and the least touch prove unbearably painful. The cutaneous reflexes are absent in the anæsthetic areas but are exaggerated in the hyperæsthetic regions. Involvement of the vegetative nervous system may be manifested by profuse sweating or by the occurrence of œdema and cutaneous vaso-

MULTIPLE NEURITIS

dilatation. Occasionally trophic changes occur in the skin, which assumes a glossy appearance, and trophic ulcers may develop. The rectal and vesical functions are practically never affected, but weakness of the abdominal muscles may cause difficulty in defæcation and micturition. Amenorrhœa frequently occurs. The cranial nerves are often involved, particularly the ocular. Optic atrophy is rare, and does not usually lead to complete loss of vision, and generally there is only a central scotoma for colour vision. The facial nerve may be affected on one or both sides. The vagus not seldom suffers, and its involvement is manifested by the increased frequency of the pulse, and occasionally by cardiac dilatation. Diaphragmatic paralysis may result from neuritis of the phrenic nerves.

The most frequent concomitant psychical disturbance of alcoholic neuritis is Korsakoff's syndrome, described in the article on **ALCOHOLISM**. The course of the disease may be acute or subacute. A very acute form, developing within a few days to the maximum degree of paralysis and accompanied by fever, frequently ends fatally within about fourteen days; it is probably due to a bacterial infection complicating the alcoholic neuritis. Occasionally the paralysis rapidly ascends from the lower extremities and resembles Landry's paralysis (q.v.). In acute alcoholic neuritis the general asthenia and the involvement of respiratory muscles makes the grave prognosis still more unfavourable. In the subacute type the paralysis may appear some days after the pains and paræsthesiæ have been noted, and may reach its culminating point only after several weeks or even months. When the development is slow an equally prolonged period frequently passes before the symptoms begin to disappear. In all these subacute cases the prognosis is good, and very rarely does any permanent paralysis ensue.

From the onset of paralysis in any muscle there is a diminution of its excitability to the faradic current; the diminution is progressive, and after a varying period the excitability disappears completely. *Pari passu* with the diminution of faradic excitability, the excitability to the constant current increases till the muscle has completely lost all faradic excitability; and then, if no recovery sets in, the galvanic excitability also diminishes, the muscle responding in a sluggish fashion to the stimulus. This stage, known as the *complete reaction of degeneration*, may persist for some

weeks. Should no recovery take place after a period of some months, the galvanic excitability finally disappears, becoming extinct about a year after the onset of the paralysis.

The treatment of alcoholic neuritis will be considered with that of multiple neuritis in general, but it may be pointed out that special care should be devoted to combating the general asthenia which forms so prominent a feature of it.

Lead neuritis.—This form of neuritis is characterized by the fact that the sensory nerves are practically never affected, and that the paralysis is, as a rule, confined to certain groups of muscles. Lead palsy occurs as an occupational disease in all workers who are brought into daily contact with raw materials containing lead. Plumbers, painters, and workers in lead glaze are particularly liable to it. It also follows the ingestion of "soft" water from lead pipes. The onset of the neuritis is generally attended by constitutional symptoms such as colic, joint and muscle pains, severe headaches, and anæmia. The well-known "blue line" in the gums is not invariably present. Nephritis and arterio-sclerosis are common in the more chronic forms of intoxication. As a rule the upper extremities alone are affected, the paralysis being limited to the extensors of the wrist, causing the characteristic wrist-drop. Occasionally one arm alone is affected, and that the one most frequently used. The distribution may be atypical, varying from a neuritis of the nerves supplying the intrinsic muscles of the hand to a generalized paralysis of all the limbs. A fibrillary tremor of the muscles is often noted. Sensation is very rarely affected, and the paræsthesia and pains so characteristic of alcoholic neuritis are generally absent. Though the cranial nerves are very seldom affected, all the lesions described under Alcoholic Neuritis may occur. The cerebral symptoms are referable to the general toxæmia. Complete recovery is the rule, but when more than one attack has occurred a permanent paralysis may be left.

Arsenical neuritis may follow chronic arsenic poisoning from the use of arsenical pigments and drugs. It has occurred epidemically from contamination of beer with arsenic. It is generally heralded by symptoms of gastrointestinal irritation. The motor nerves of all four limbs are, as a rule, affected. The sensory disturbances may resemble those of alcoholic neuritis; loss of muscle and joint sensation is a common feature. The prognosis is generally

good as regards life, but permanent paralysis often results.

Diphtheritic neuritis, the commonest of the forms due to bacterial toxins, is fully described in the article on DIPHTHERIA (q.v.). The chief danger in this form of neuritis is the affection of the vagus, leading to acute dilatation of the heart and cardiac failure. Occasionally aspiration pneumonia results from the difficulty in swallowing and the anæsthetic condition of the epiglottis. When the respiratory muscles are paralysed, death may take place from asphyxia. The paralysis of the skeletal muscles disappears very slowly, but permanent paralysis is rare. Once diphtheritic neuritis has set in, there is no reason to think that serum treatment has any effect. Antitoxin treatment of diphtheria cannot be said to be of much service in preventing the subsequent development of neuritis, but it seems probable that if more vigorous treatment of the local throat lesion were practised the incidence of diphtheritic neuritis would be greatly diminished.

The **differential diagnosis** of multiple neuritis, as a rule, offers little difficulty. The non-segmental distribution of the paralysis, its flaccid form, the absence of the deep reflexes, the fact that the plantar response remains flexor when the foot muscles are not so paralysed as to abolish it, the tenderness of the nerves, and the absence of sphincter trouble, differentiate it from lesions of the cerebro-spinal axis. When anæsthesia is absent some difficulty may be experienced in distinguishing it from *anterior poliomyelitis*, especially when the case is seen long after the onset. The development of the neuritis is, however, always much slower than that of poliomyelitis, and there is generally some loss of muscle and joint sensibility.

Treatment.—The specific treatment of the causal agencies of multiple neuritis will be found under their several headings. The main indications in the treatment of the neuritis itself are to preserve the paralysed muscles in as good a condition as possible till motor innervation is restored, and to prevent the occurrence of postural contractures. The first of these ends is attained by the daily stimulation of muscular contraction by electricity and by kneading of muscles to promote circulation of the lymph; but such treatment must be delayed till the initial tenderness of the nerves and muscles has abated. The second object is achieved by the daily performance of passive

movements, and by the use of simple apparatus consisting of light jointed splinting coupled up by elastic cords, that can neutralize the overaction of unparalysed antagonistic muscles. The occurrence of contracture is a reproach to those in charge of the case. Electrical or manipulative treatment of the affected nerves is useless and may often do harm. Full doses of aspirin generally prove efficient for the relief of pain; morphia is rarely required. It is the fashion for both patients and practitioners to ascribe many of the vague pains felt by elderly and arterio-sclerotic patients to neuritis, and though the pathology of these pains is obscure there is something to be said for the diagnosis on purely clinical grounds. Dietetic treatment, with aspirin as an analgesic, purgation, and the use of mild doses of diuretic drugs, often gives good results in these cases.

F. L. GOLLA.

MUMPS (*syn.* Epidemic or Specific Parotitis).—An acute contagious disease, characterized by an inflammation of the parotid and to a less extent of the other salivary glands.

Etiology.—The specific organism has not been isolated. The work of Mervyn Gordon suggests that it belongs to one of the filterable viruses. This observer inoculated ten monkeys with an intracerebral injection of a filtrate of saliva from children in the acute stage of mumps. Four of the animals died, one developed an illness from which it recovered, and five were not affected. The symptoms, which were those of meningeal irritation, developed one to two days after injection. The principal lesions observed post mortem were a varying degree of lymphocytic meningitis, and an acute interstitial parotitis in the animal which survived longest. More recently, Martha Wollstein has found that, by injecting the parotid gland and testicle of cats with a sterile filtrate of the saliva of children and adults in the active stages of mumps, a condition similar to that of mumps in human beings resulted—a rise of temperature, swelling of the parotid and testis, and polymorphonuclear leucocytosis being noted after an incubation period of five to eight days. The virus was most readily detected in the saliva during the first three days of the disease, was less readily found on the sixth day, and was entirely absent after the ninth day. The virus was also detected in the blood of patients showing severe constitutional symptoms. The serum of recovered cats was found to contain an immune body

which diminished or even neutralized the action of the virus of mumps.

Infection is probably conveyed from the mouth to the glands by the salivary ducts. The disease is transmitted by direct contact, and is not conveyed through the air for any distance. Its infectivity commences in the prodromal stage, and usually ceases a week after the subsidence of the parotid swelling.

Mumps is rare in infancy, most frequent between 5 and 15 years, and not uncommon in early adult life. Males are chiefly affected. The disease, prevalent in endemic or epidemic form in all parts of the globe, is commonest in the spring and autumn. One attack usually conveys immunity for the rest of life.

Pathology.—The characteristic anatomical lesion is an acute interstitial parotitis. The interacinous and periglandular tissue shows a sero-fibrinous exudate, while the glandular tissue itself is less affected. Lymphocytosis, absolute and relative, is a constant and early feature, and usually lasts till the parotid swelling has subsided. The eosinophils are slightly decreased at the onset, but the basophils are unaffected (Wile). Orchitis is sometimes accompanied by an increase in the polymorphonuclears.

Symptomatology. The incubation period is usually 18–21 days, but may be as short as 8 or as long as 26 days. A prodromal period of one or more days may occur, characterized by fever, headache, vomiting, or convulsions; but usually the first symptom, especially in children, is a slight fullness of the parotid gland, first noticed in the space between the mastoid process and the ascending ramus of the lower jaw.

Older patients complain of local discomfort and earache, especially on opening the mouth, on mastication, or on ingestion of acids. The swelling is often exquisitely tender. Except in severe cases, the temperature does not rise above 102° F., and there is little constitutional disturbance. The swelling increases for two or three days, and lasts from a few days to a fortnight, according to the severity of the attack. It is frequently associated with some submaxillary and sublingual involvement, causing considerable facial deformity. Inflammation of either of these glands apart from the parotid may occur. The skin of the affected area may be almost normal in appearance, or shiny, tense, and traversed by prominent veins.

The affection may be entirely unilateral, in which case the left side is most frequently

involved, but usually the opposite gland becomes inflamed two or three days after its fellow. Sometimes the interval may extend to five or six weeks. Complete resolution is almost invariable; suppuration or sclerosis is very rare. Salivary secretion is usually diminished. Sialorrhœa is rare. Some facial or palatal injection is not uncommon, but the severe tonsillitis described by French writers is exceptional.

Complications.—**Orchitis**, the most important complication, seldom occurs in patients who have not attained puberty, and usually not until the parotitis has subsided. It may, however, be a prodromal symptom or even the solitary manifestation of the disease (orchitis parotidea). (See ORCHITIS.) In women the breasts, ovaries, and labia majora may be similarly though much less frequently affected.

Pancreatitis is another complication which is more frequent in adults than in children. The symptoms are pain and tenderness in the epigastrium, nausea and vomiting, constipation or diarrhœa. Acetone and diacetic acid may be found in the urine. As a rule the complication is of short duration and good prognosis, but cases of diabetes as a sequel are on record. Probably some of the cases described as pancreatitis following mumps are really examples of gastritis (Cheinisse).

Meningitis.—Cerebro-spinal lymphocytosis is not uncommon in mumps. Meningeal symptoms may occur at any period of the disease, but are usually ill-marked, and consist only of vomiting, headache, slight nuchal rigidity, rise of temperature, and a slow pulse. The complete picture of meningitis is much less common. Roux found only three examples in 274 cases of mumps. The cerebro-spinal fluid is usually clear and sterile under hypertension, and shows more or less lymphocytosis. Purulent meningitis due to streptococci and staphylococci has been reported by Perrin.

Encephalitis may be a sequel of mumps as of other infectious diseases. The onset is characterized by convulsions followed by coma and flaccid paralysis or contracture of the limbs. Transient or permanent hemiplegia may occur, accompanied by aphasia, or the latter may be an isolated symptom. **Psychoses** such as mania or melancholia have also been recorded. Rarer nervous complications are facial paralysis, acute bulbar palsy, poliomyelitis, and herpes zoster.

The **special senses** are sometimes involved. The most serious complication of this kind is

deafness, due to non-suppurative disease of the labyrinth. Three forms have been described, according as the cochlea, the semicircular canals, or the labyrinth as a whole are affected. As a rule only one ear is attacked, but the condition may be bilateral. The onset may be insidious or associated with dizziness, vomiting, and tinnitus. The deafness is usually permanent and resists all treatment. Otitis media may also occur.

Several ocular complications have been reported, e.g. conjunctivitis, keratitis, and iritis. Inflammation of the lachrymal gland may accompany parotitis, or may even be the only manifestation of mumps (Tronzo). The most serious ocular complication is optic neuritis, which often ends in atrophy.

Ciliary palsy and paralysis of the external and internal rectus have been recorded after mumps, but have probably been due to an overlooked diphtheria.

Apart from bradycardia, which is a symptom of meningeal involvement, the heart is rarely affected in mumps. A few cases of myocarditis and pericarditis have been recorded, as well as of endocarditis both of the mitral and of the aortic valves.

Enlargement of the spleen as a manifestation of mumps has recently been described by Capitan, who found it almost invariably present in 700 cases of mumps in soldiers.

Arthritis similar to that occurring in scarlet fever may be met with. As a rule, it occurs as arthralgia without any local swelling, but in rare cases there is visible swelling of the affected joints. Resolution as a rule takes place rapidly; suppurative arthritis is extremely rare.

Slight **albuminuria** is not uncommon, but hæmaturia and other symptoms of nephritis are exceptional.

Other rare complications of mumps are prostatitis, urethritis, and vesiculitis accompanying the orchitis, thyroiditis, oedema of the glottis—sufficiently severe to require tracheotomy—pleurisy and pneumonia.

Diagnosis.—Mumps must be distinguished from the parotitis secondary to other diseases, especially to diphtheria, typhoid fever, pneumonia, and scarlet fever. The facial deformity of toxic diphtheria is particularly misleading, and this condition is often mistaken for mumps. Inspection of the throat is therefore imperative before making a diagnosis of mumps, especially in children.

Parotitis may also follow operations on the

abdomen or pelvis, or complicate septic processes in those regions, apart from any surgical interference. It has also been known to occur in poisoning by lead, mercury, iodine, and by mustard gas in warfare.

It is noteworthy that secondary parotitis, from whatever cause, is much more likely than mumps to end in suppuration.

Mumps is to be distinguished from cervical adenitis by its more sudden onset, its localization, and shorter duration.

Prognosis.—The prognosis is generally good. The dangers lie in the permanent disablement which may be caused by some of the complications. Testicular atrophy, if bilateral, will cause sterility. Otitis may also be bilateral and result in complete and incurable deafness. Encephalitis may produce hemiplegia, and pancreatitis has been followed by diabetes. All these complications, however, are very uncommon. They are more likely to occur in adults than in children, and in males more than in females.

Treatment.—In most cases little is required beyond keeping the patient in bed, and the administration of a fluid diet. If the pain be severe, glycerin-of-belladonna fomentations will afford relief. Prevention of oral sepsis by attention to the teeth and gums should be enforced. Rest in bed is probably the best prophylactic against orchitis, but, if this be too irksome, adult males on getting up should wear a suspensory bandage and should be forbidden to take part in any violent exercise for some weeks. On the occurrence of orchitis a purge should be given and opium or belladonna applications ordered. The pain of pancreatitis should be treated by hot fomentations to the epigastrium. Morphia will rarely be required. Meningeal symptoms can usually be relieved by lumbar puncture.

J. D. ROLLESTON.

MURMURS (see HEART, FUNCTIONAL MURMURS OF; and VALVULAR DISEASE, CHRONIC).

MUSCÆ VOLITANTES (see VITREOUS, AFFECTIONS OF).

MUSCLES, INFLAMMATION OF (see MYOSITIS).

MUSCLES, PAIN IN (see MYALGIA).

MUSCLES, SPASM OF (see CRAMP).

MUSCLES, TUMOURS OF (see MYOMATA).

MUSCULAR ATROPHY, DIAGNOSIS OF

MUSCULAR ATROPHY, DIAGNOSIS OF.

Atrophy of muscle may result from disease primarily involving the muscle-fibre itself, or from a lesion of the motor neurone which innervates it. An additional important cause of muscular wasting is arthritis, for example of the shoulder, hip, or knee. This also has its characteristics by which it may be recognized. For purposes of diagnosis, therefore, cases of muscular wasting fall into four main groups, viz. (1) primary myopathy and allied primary muscular diseases (see MYOPATHY; MYOTONIA ATROPHICA; MYOTONIA CONGENITA); (2) progressive muscular atrophy due to degeneration of the lower motor neurones in the cord and brain-stem (see MUSCULAR ATROPHY, PROGRESSIVE); (3) muscular atrophy following paralysis of nervous origin; and, finally, (4) atrophy following a local arthritis. In the first, muscular wasting constitutes the disease; in the others, it is merely symptomatic of disease of the nervous system, and varies in all its characters according to the nervous lesion which underlies it.

The differential diagnosis of the various clinical conditions in which these types of muscular atrophy are found depends on the mode of evolution and the topography of the wasting, on the presence of certain characteristics of the wasting muscles, on the coexistence of other signs of disease of the nervous system, and on the presence or absence of familial and hereditary factors.

In the primary muscular diseases, or primary myopathies, wasting is the essential lesion, of which the weakness that ensues is the result; but in the secondary muscular atrophies this is not always the case. Arthritic atrophy is related to the joint affected. Speaking generally, myopathy and progressive muscular atrophy are characterized by a slowly progressive onset of wasting and associated weakness, while wasting due to paralysis is characterized by a rapid onset of loss of power followed by a slow wasting. Arthritic wasting is rapid in onset but soon reaches a stationary condition. It is with the first two groups that we are mainly concerned here. The differentiation of these may be considered under the following headings:—

Topography.—Bilateral symmetry of wasting is of the greatest diagnostic importance, and is characteristic of the various clinical forms of primary myopathy. In progressive muscular atrophy of spinal-cord origin, the wasting, though bilateral, is seldom simulta-

neous in onset on the two sides, and hence is usually more advanced in one upper limb than in its fellow. This is also true of syringomyelia, which presents a similar clinical picture of muscular wasting. A unilateral wasting of the intrinsic muscles of the hands which does not become bilateral may be due to a cervical rib. Similarly, unilateral wasting round a shoulder-joint may follow an acute arthritis of that joint.

Of equal importance is the distribution of the wasting throughout the musculature. In general, it may be said that in myopathy the muscles of the limb girdles and of the roots of the limbs are earliest, most profoundly, and often exclusively affected. The distal limb-muscles remain immune throughout. Muscular atrophy of spinal-cord origin, on the other hand, involves, as a rule, the intrinsic hand and forearm muscles earliest and most severely, though the upper arm, shoulder, and neck muscles are eventually affected. This law is not without exception, for a certain small number of cases of progressive muscular atrophy show an initial involvement of muscles round the shoulder, while in the rare distal type of myopathy, wasting begins below the knees and elbows. It is upon associated physical signs that we depend for differentiation in these exceptional instances.

Commonly, too, in peripheral neuritis, and in lesions of such nerves as the median and ulnar, the small hand muscles are more profoundly affected than the more proximal muscles of the forearm, but here, again, differentiation is readily attained owing to the presence of other characteristic signs.

In progressive muscular atrophy, in addition to the affection of the distal limb-muscles, certain muscles of the trunk and face are involved, particularly the intercostals and diaphragm, and those of the tongue and soft palate, and the muscles of phonation and deglutition. In myopathy, on the other hand, the muscles of respiration, deglutition, and phonation and those of the tongue are always immune, and the characteristic symptomatology of bulbar paralysis is not seen, the facial muscles affected being the orbiculares, zygomatici, and the masseters.

Fibrillation.—In myopathy, fibrillation is extremely rare, and for all practical purposes may be said not to occur. In peroneal muscular atrophy it is occasionally seen, but it is in progressive muscular atrophy that fibrillation is most marked. Spontaneous flickering

contractions are common, and when not present are readily induced by passive stretching of a muscle. It is not constant, however, nor confined to this disease; after certain debilitating illnesses there may be widespread fibrillation ("myokymia").

In myopathy, and to a less degree in peroneal muscular atrophy, the direct mechanical irritability is early and greatly diminished, while in progressive muscular atrophy it is, and remains to a late stage, increased.

The tendon reflexes of the affected muscles diminish and finally disappear in myopathy as wasting proceeds, but, owing to the presence of lateral-column degeneration, in progressive muscular atrophy (amyotrophic lateral sclerosis) both the knee- and ankle-jerks are strikingly increased, and there may even be clonus.

The state of the **electrical reactions** may also afford information of diagnostic importance. In the primary myopathies the excitability of the muscles to both the continuous and the faradic currents diminishes progressively and finally disappears without undergoing that qualitative change known as the reaction of degeneration. In progressive muscular atrophy, on the other hand, during the period of active wasting this reaction may be present. It is also to be found in muscular atrophy following paralysis from disease of peripheral nerves.

The association of wasting of certain muscles with enlargement, or pseudohypertrophy, of others is found only in myopathy. In these cases the incidence of both changes is pathognomonic of this disease. The general rule is that in the secondary muscular atrophies the power of the muscles is fair in relation to their size, while in the myopathies their strength is much less than would be anticipated from their bulk.

Contracture occurs in both maladies, but most extensively in myopathy, in which lordosis, curvature of the spine, and extensive deformities of the limbs are seen. Early talipes equinus is almost pathognomonic of peroneal muscular atrophy; while in muscular atrophy due to lesions in the cervical enlargement of the cord, claw-hand is the usual deformity found.

The presence of **myotonia**, or tonic spasm and delayed relaxation of muscles on voluntary effort, is found only in those rare diseases of muscles, Thomsen's disease and myotonia atrophica, and is pathognomonic of them.

Other signs of disease of the nervous system

are of great importance in distinguishing wasting of nervous origin from the primary myopathies. Thus, degeneration of the pyramidal tracts, manifested clinically as spastic paresis of the lower limbs with increased tendon reflexes and the extensor type of plantar response, is suggestive of that form of progressive muscular atrophy known as amyotrophic lateral sclerosis. This in turn has to be distinguished from such lesions of the cervical segments of the cord as hypertrophic pachymeningitis, or syringomyelia. The presence of root-pains, of sensory loss, of irregular distribution, and of other signs of syphilis of the nervous system, differentiates the former, while the characteristic sensory changes and the arthropathies render the diagnosis of syringomyelia relatively easy. In muscular atrophy due to local or generalized peripheral-nerve diseases, the evolution of the symptoms—the initial weakness with the later wasting, the great pain and tenderness of the muscles, and the sensory changes—is sufficient to make a differential diagnosis.

The characteristic distribution, bilateral symmetry and slow onset of bulbar paralysis separates this disease from other bulbar lesions, while the presence of wasting and fibrillation of the tongue and the absence of pyramidal-tract involvement mark it off from pseudobulbar paralysis.

Muscular atrophy secondary to arthritis is characterized by its rapid onset and early development to a stationary condition, by the absence of fibrillation and of the reaction of degeneration, and by the presence of increased reflex and mechanical irritability.

Finally, from the age of onset we may obtain valuable information as to the nature of the wasting. Myopathy begins during infancy or childhood, progressive muscular atrophy always in adult life. A familial or hereditary incidence is also characteristic of primary myopathy. Progressive muscular atrophy and other atrophies of nervous origin do not present this feature.

F. M. R. WALSHE.

MUSCULAR ATROPHY, PERONEAL

(Neuritic or Charcot-Marie-Tooth Type).—A form of progressive muscular wasting allied to both myopathy and the spinal muscular atrophies. Clinically it consists in wasting and weakness of the distal muscles of the limbs, while anatomically there are found changes in the spinal cord. It is familial and hereditary,

affects and is transmitted by both sexes, and develops during childhood. It often appears after an acute febrile illness such as measles. On pathological examination, sclerosis of the posterior columns of the cord and atrophic changes in the cells of the ventral horns and of Clarke's columns are found. The nerve-fibres in the affected muscles are degenerated and the muscular fibres are atrophic.

Symptomatology.—The onset is usually during the first decade of life, but may be later. Wasting first appears in the small muscles of the foot, and then in the peronei and the long extensors of the toes. This incidence of weakness produces talipes equino-varus, and thus early club-foot is a characteristic symptom of the disease. Later, the calf and thigh muscles become involved. The wasting first invades the distal portion of an affected muscle, spreading in a proximal direction throughout the whole muscle. When the muscles of the lower third of the thigh have been thus affected, the limb assumes a curious inverted Indian-club form. Some years later the upper limbs become involved, first the small hand muscles and then those of the forearm. Claw-hand develops, and there may also be wrist-drop from involvement of the extensor group. The condition is usually, though not constantly, bilaterally symmetrical.

To both the continuous and the faradic currents there is progressive quantitative diminution of excitability in the muscles; the reaction of degeneration is rare. The muscles occasionally fibrillate. They are painless and not tender.

The muscles of the head and neck and those of the trunk are unaffected. The sphincters are also intact. In a certain proportion of cases there is impairment of all forms of cutaneous sensation overlying the wasted muscles. The ankle-jerks disappear early, and the knee-jerks follow when the vasti are affected. The cutaneous reflexes are unaltered.

The disease is so slowly progressive that patients remain active well into middle life, and the termination is due to some intercurrent affection. Recovery is not known. Treatment is unavailing. The foot-drop may require some mechanical support.

F. M. R. WALSHE.

MUSCULAR ATROPHY, PROGRESSIVE (Chronic Poliomyelitis, Bulbar Paralysis, Amyotrophic Lateral Sclerosis).—A disease characterized by progressive degeneration of the motor cells of the ventral horns of the

spinal cord, and of the cells of certain motor nerve nuclei in the brain-stem. With it is usually associated a descending degeneration of the pyramidal tracts; that is, there is degeneration of both upper and lower motor neurones. The principal incidence of these pathological changes varies in different instances, giving rise to variations in the clinical features of the disease, and thus three well-defined clinical types emerge: The first is *progressive muscular atrophy*, in which degeneration is confined to the ventral-horn cells of the cord, commonly to those of the cervical segments. This produces a slowly progressive atrophy and weakness of the distal muscles of the upper limbs of the Aran-Duchenne type. Much less frequently the lumbar enlargement of the cord is similarly affected, producing wasting and weakness of the lower limbs. Such a pure lower-motor-neurone affection is, however, comparatively rare, and the second clinical type, *amyotrophic lateral sclerosis*, in which the degeneration of the cells of the ventral horns is associated with degeneration of the pyramidal tracts, is much more frequently observed. Clinically this type is characterized by atrophic paralysis of the upper limbs of the Aran-Duchenne type with spastic paresis of the lower limbs. In the third type, *bulbar paralysis*, the nuclei of certain motor cranial nerves are profoundly, but not always exclusively affected, producing the characteristic clinical picture of this condition, associated, it may be, with progressive muscular atrophy of spinal type, or with amyotrophic lateral sclerosis. Chronic poliomyelitis is considered with the lower-motor-neurone type of the disease, and has no separate identity.

This classification will be followed, but it is emphasized that in these three symptom-complexes we are dealing with what is essentially a single disease.

Etiology.—The cause of the disease is obscure. Cold, trauma, exposure, strain, and developmental defect have all been invoked. Lead and syphilis have also been held responsible for certain cases. Undoubtedly syphilis holds a definite place in the etiology of one group of cases of the amyotrophic lateral sclerosis type, in which a meningo-myelitis is the cause of the symptoms, and these frequently present clinical features differentiating them from the ordinary cases of non-syphilitic origin, as the presence of other signs of syphilis of the nervous system, such as the Argyll-Robertson pupil, miosis and irregularity of the pupils,

MUSCULAR ATROPHY, PROGRESSIVE

sensory and sphincter defects, and changes in the cerebro-spinal fluid.

The disease appears in adult life during the third and fourth decades, and is more frequently seen in males. It is neither a familial nor an hereditary disease.

Pathology.—The essential underlying change in the nervous system consists in an intense primary degeneration in the ventral-horn cells of the cervical segments of the cord. The nuclei of the seventh, eleventh, and twelfth cranial nerves also show similar changes in cases with bulbar symptoms. There is, even in those rare cases which run their course clinically as pure progressive muscular atrophy, degeneration of the pyramidal tract throughout its course, including its cells of origin in the cerebral cortex. This degeneration is most intense and of longest standing in the lower segments of the cord. There is also diffuse degeneration of the whole ventro-lateral columns, but of this, apart from signs of affection of the pyramidal tracts, there is no clinical evidence. Some secondary neuroglial proliferation may accompany these primary changes.

Symptomatology. Progressive muscular atrophy.—The symptoms consist in wasting and weakness of the muscles supplied by the motor fibres that take origin from the degenerating cells. The arms, as a rule, are first affected. One limb is usually attacked earlier than its fellow. An aching pain in the small hand muscles may precede visible wasting, which comes on with weakness in the thenar muscles and interossei. The thenar eminence flattens, the interosseous spaces become hollowed, and the metacarpal bones exposed beneath the skin. The forearm muscles, and then the biceps, triceps, and deltoid, are successively involved. The back and shoulder girdle muscles are early affected in rapidly progressive cases. The lower half of the trapezius, the rhomboids, and erector spinæ waste, then the sterno-mastoids and the extensors of the neck. This results in rotation and winging of the scapulae and inability to hold erect the head, which tends to fall forwards or backwards. Eventually the intercostals, pectorals, and latissimus dorsi are involved and the consequent respiratory difficulty may terminate the illness.

The wasting may be extreme in the muscles affected, the limbs become mere skin and bone, and deformities such as claw-hand occur. Less commonly the lower limbs are similarly affected

and waste, the glutei and the extensors of the knee being, as a rule, the most severely wasted.

Fibrillation is a very prominent feature in the wasting muscles, and widespread spontaneous flickering contractions may be seen. The mechanical irritability of the muscles is also greatly increased, and remains so as long as there is contractile muscle substance left.

The reaction of degeneration may be found in the affected muscles, and the tendon-reflexes which depend on the muscles involved in this atrophic paralysis are abolished. Sensory changes are absent, and the sphincters are intact. The interval between the affection of the two arms varies, and may be longer than a year. The progress of the disease also varies in rapidity and extent. In some cases an apparent arrest occurs before the respiratory muscles are affected, and for years the disease may be limited to the arms. In other cases the spread to the neck, trunk, and respiratory muscles is more rapid, bulbar symptoms may be super-added, and death results from some pulmonary complication.

Bulbar paralysis.—This is the expression of a degeneration of the cells of the motor cranial nerve nuclei in the medulla, similar to that found in the cervical segments of the cord in progressive muscular atrophy. It occurs chiefly in males during the second half of life. From its topography it is also known as labio-glossolaryngeal paralysis. The onset is gradual and the course slow.

The tongue is earliest and most severely affected. An indistinctness of speech, due to slurring of the lingual consonants, is usually the first symptom, and may, during the early stages, appear only in fatigue. The mobility of the tongue and lips then becomes deficient, pronunciation and the power of whistling are impaired, the soft palate and the pharyngeal muscles fail slowly so that the voice weakens owing to the impairment of articulation, fluids regurgitate through the nose, and there is difficulty in swallowing solids. These defects are aggravated by the weakness of the lips, which allows the dribbling of food and saliva from the mouth. Sooner or later the muscles of the larynx are involved, and in some cases may be more severely affected than those of the pharynx, so that clinically we see either deglutition or phonation predominantly impaired. When the latter is the case the patient develops an ineffectual cough, and particles of food are apt to enter the glottis, causing aspiration

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pneumonia, which, indeed, is a frequent termination of the illness.

The chief objective signs are atrophy, wrinkling of the mucosa and fibrillation in the tongue, and wasting of the lips. In the later stages articulation is quite lost, and the patient is able to utter only feeble, low-pitched, inarticulate sounds. Deglutition may become impossible, and the patient presents a pitiable spectacle, with half-opened dribbling mouth, expressionless face, and often great emotional instability.

This symptom-complex may exist alone, or in combination with a progressive muscular atrophy of the Aran-Duchenne type. In the latter case the bulbar affection may either be a late complication or may usher in the disease.

Considerable general weakness and emaciation result from this condition, on account of the extreme difficulty of taking adequate nourishment, while bronchitis and pneumonia may follow the aspiration of food particles into the larynx and trachea. These complications, together with the respiratory difficulty due to affection of respiratory muscles, are the common cause of death in this disease.

Pseudo-bulbar palsy is considered under that title.

Amiotrophic lateral sclerosis.—The clinical characteristics of this type are flaccid wasting and weakness of the upper limbs, of the distribution described in pure progressive muscular atrophy, with spastic paresis of the lower limbs. In addition, therefore, to the lesion of the ventral horns underlying the wasting of the arms, there is degeneration of the pyramidal tracts.

This is the most frequently observed form of the disease. The lateral sclerosis, and therefore the spastic weakness of the legs, may precede the atrophy of the arms, or may appear later. It is probable that most cases of so-called primary lateral sclerosis are of the former class.

Although the lower limbs are most constantly and most severely affected by this lesion of the upper motor neurones, yet, since the whole cortico-spinal path is involved, symptoms of pyramidal-system affection elsewhere may be seen in such phenomena as increased jaw-jerk or jaw-clonus, and signs of spastic weakness of the upper limbs. The spastic weakness of the lower limbs is of the type seen in the leg in residual hemiplegia—that is, the limbs are rigidly extended and adducted, the knee- and ankle-jerks are increased, and there may be

clonus. The abdominal and cremasteric reflexes are abolished, while the plantar reflex is of the extensor or Babinski type.

There is no sensory defect, and the sphincters are intact.

Prognosis.—The course of the disease in all its clinical forms is steadily progressive. Arrest is rare, and when seen is usually so late in the course of the malady that the patient is already disabled. In some cases the evolution and course are rapid from the onset, and the patient may reach the terminal stages within six to twelve months, death occurring from some pulmonary complication. On the other hand, the progress may be slow and limited in extent, and in these cases the patient may live for years. Recovery does not occur.

Treatment.—The course of the disease is not profoundly influenced by any form of treatment. The best results are obtained from careful active and passive exercise. The latter is best given as massage and passive movements of the limbs. Moderate active exercise, short of fatigue, is also of the greatest importance. It is doubtful if electrical treatment has any value other than as a form of gentle exercise of muscles. Weak galvanism, just sufficient to produce contraction, and applied by a roller electrode over the affected muscles, affords the best means of providing this.

The maintenance of the general health by the use of cod-liver oil and such drugs as iron and arsenic is also indicated. When bulbar symptoms occur, the nutrition of the patient calls for special care. Semi-solids are more easily swallowed than either liquid or solid food. When deglutition is lost, the use of an œsophageal tube may be necessary. Rectal feeding is valuable as an adjuvant to this, but alone is inadequate.

Gowers advocated as a means of arrest of the disease, and as productive of actual improvement, the use of hypodermic injections of strychnine nitrate in doses of $\frac{1}{16}$ gr. daily. This may be increased progressively to $\frac{1}{8}$ gr. The treatment may be continued indefinitely. In early cases of rapid onset when there is reason to suspect the syphilitic origin of the disease, antisyphilitic treatment may be carried out. Mercurial inunction combined with small weekly doses of salvarsan for a period of six weeks may produce striking arrest and improvement of the condition.

F. M. R. WALSH.

MUSCULAR DYSTROPHY (see MYOPATHY).

MYALGIA

MUSCULAR RHEUMATISM (see MYALGIA).

MUSCULO-CUTANEOUS PARALYSIS (see SPINAL NERVES, LESIONS OF).

MUSCULO-SPIRAL PARALYSIS (see SPINAL NERVES, LESIONS OF).

MUTISM (see HYSTERIA).

MYALGIA.—Of all the afflictions encountered in general practice, none is more common than those which are here grouped together under the term myalgia. The title "fibrositis," sometimes used to describe these disorders, is not employed here because it is also applicable to conditions other than those about to be noticed. Another term, muscular rheumatism, carries unwarranted and probably untrue implications as to etiology. Other titles used to describe particular localizations of the disorder are lumbago (q.v.), pleurodynia, omodyn timer, etc., while the conditions described as brachial neuritis and rheumatic torticollis (see TORTICOLLIS AND WRYNECK) probably own a similar etiology.

Etiology.—Among *predisposing* factors, sex is important; myalgia is far commoner in men than in women. It is a disorder of the middle and later periods of life, though in children the sterno-mastoid and in adolescents the intercostal muscles are often attacked. There is an hereditary liability to myalgia, probably metabolic, since it is closely connected with the gouty diathesis. Exposure to wet, especially if combined with cold, is an important causal factor; hence it is that certain outdoor occupations involve a liability to myalgia. So also do pursuits in which there is frequent exposure to sudden changes of temperature. In other occupations, frequent strain of certain muscles, such as those of the lumbar region, predisposes to myalgic attacks. Not only is myalgia provoked by chronic injury of this type; it may also be suddenly precipitated by some acute stress of the muscle attacked, especially if a predisposition is present. Lastly, the potent effect of fatigue must be noticed, particularly in relation to the brachialgic forms.

The *exciting* causes are unknown. There is some reason, however, for accusing the process known as subinfection—that is, the organisms normally inhabiting the alimentary and respiratory tracts escape into the system, exercising a brief pathogenic action on the tissues to which they find their own way.

It is certain also that whatever causes articular gout is also responsible for many cases of myalgia.

Pathology.—There is reason to suppose that the essential process is inflammation of the fibrous investments of the skeletal muscles, with their extensions to neighbouring tissues such as nerve-sheaths, bursae, etc. Probably the reaction is particularly concentrated in certain spots, so that inflammatory nodules are formed, from which small masses of scar-tissue develop.

Symptomatology.—Pain leading to inhibition of movement is the characteristic symptom. In acute cases it is intense; in the chronic forms it is more of an ache than an actual pain. Often the easing of the acute muscular phase is followed by a chronic neuralgic period. The pain is associated with tenderness of the affected muscles, sharp in acute cases, but of little or no importance in chronic cases. Active movements aggravate the pain; so to a less extent do passive movements, especially if they put the inflamed muscle on the stretch. Reflex hypertonus, and possibly inflammatory congestion, cause the muscle to feel tenser than normal. Indurations and nodules, firm and hypersensitive, are to be felt in the inflamed area, the frequency with which they are detected varying rather widely with the skill and prejudices of the observer.

Diagnosis. The difficulty is to distinguish between mere myalgic pain and lesions of neighbouring bones, joints, or viscera; to separate between pleurodynia and pleurisy, between lumbago and lumbar caries, and so on. But in true myalgia there is deep muscular tenderness as a rule, in the acute phases at all events, while in visceral or bony disease the appropriate physical signs will be detected sooner or later.

Prognosis.—The usual course is towards recovery. Acute attacks may clear up in a few days, or may resolve up to a certain point, leaving behind them aches, probably due to perineuritis, which linger for weeks or months. The more severe the original attack the greater the liability to long after-pains. Even after recovery, relapse is particularly likely to occur. In a few cases the pain becomes practically continuous and permanent, crippling the victim and making his life a burden.

Treatment.—The first indication in an acute phase is to secure rest for the affected part. For patients whose trunk muscles are

painful, bed is the best place, for a day or two at any rate. For severe pleurodynia the side should be strapped. Pain is also relieved by the application of heat or counter-irritants, or both; the turpentine stupe combines them effectively, the flat-iron applied through brown paper is a homely but efficient form of heat, while mustard in poultice or plaster is perhaps the best counter-irritant. Various local analgesics are also used, such as menthol dissolved in olive oil, oil of wintergreen in a similar solution, or aconite liniment alone or in combination with belladonna liniment and chloroform. By mouth, also, analgesics such as aspirin or pyramidon do good in most cases, but the pain—especially in pleurodynia—may be so severe as to call for injection of morphia. If there is a gouty element in the case, colchicum and alkalis should be given, and abstinence from meat, extractives, and alcohol insisted upon.

These measures reduce the pain in a few days to the level of a persistent grumbling ache; when the shoulder muscles have been affected, this often takes the form of sharp neuralgic pain down the arm, and in cases of lumbago it may become permanent, especially in elderly subjects. To terminate this sequela the causes must be carefully looked for. If the patient, either generally or in respect of any particular group of muscles, is overworked, this must be prohibited. The gouty element should be treated, also any foci of infection that can be discovered. Autogenous vaccines are said to be useful. Even in the chronic phases, mild but constant counter-irritation is often of use. Herein, probably, lies the indubitable value of the popular belladonna plaster.

In obstinate cases a visit to a spa may be recommended. As to the indications for physiotherapy, there is much difference of opinion, and it can only be said that no sharply painful muscle should be subjected to any but gentle manipulations; that as the pain becomes less severe, the scope of the movements permitted should be increased, and that it is as counter-irritants that most of the electrical and thermal appliances do their good.

Recurrences are to be prevented by extirpation of infective foci, by anti-gout treatment, by protection from cold and wet, and, above all, by avoidance of fatigue, especially of any one group of muscles.

CAREY COOMBS.

MYASTHENIA GRAVIS (*syn.* Pseudo-paralytic Myasthenia Gravis; Asthenic Bulbar Palsy).—This disease has been generally recognized in the British Isles only during the last twenty years.

Etiology.—Myasthenia may attack persons of any age and of either sex, but has no familial tendency. Although no specific cause has been discovered, it is remarkable that women suffering from the disease may lose all symptoms during pregnancy—a fact which suggests that some abnormality of internal secretion is an important causative factor.

Morbid anatomy.—Autopsies have revealed (1) the frequent presence of an enlarged thymus gland, which may show degenerative changes; (2) numerous "lymphorrhages," or collections of lymphocytes, scattered in various tissues, especially the muscles; (3) slight degenerative changes in muscle-fibres, and (4) the absence of definite abnormalities in the central or peripheral nervous systems.

Symptomatology.—Myasthenia gravis is marked by the presence of a muscular feebleness which may vary, in different patients and in the same patient at different times, from a condition of rapid exhaustibility to one of complete paralysis. This symptom may be limited to a few muscles—for instance, those supplied by the cranial nerves—or may affect the skeletal musculature as a whole. It is usually accompanied by a corresponding graduated failure of response of the affected muscles to continuous faradism, although they retain their excitability to the make-and-break of a galvanic current (*myasthenic reaction*).

The patient often complains that although he is able to do certain things in the morning, this ability disappears in the course of the day. He may be able to keep his eyes normally open for a short time, but sooner or later the upper lids droop so that he can only look straight in front of him by throwing his head back. Diplopia, due to weakness of one or more ocular muscles, is often a troublesome symptom, and difficulty in mastication, articulation, and deglutition is not uncommon. A myasthenic facies in which the smile is "sour," owing to weakness of the zygomatic and risorius muscles, has been described. The limbs may be affected in a similar manner, but the most important and most ominous complaint is shortness of breath. A paroxysm of dyspnoea, due to failure of the respiratory muscles, is a very distressing complication and the most frequent cause of death in fatal cases. The

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presence of an enlarged thymus gland can sometimes be demonstrated by percussion or by radiography during life.

The tendon-reflexes are generally normal in character, but may be easily exhausted. The superficial reflexes are unaltered. Control over the sphincters is not interfered with, although in one or two cases slight incontinence of urine has been recorded. Sensory disturbances are not very frequent, but patients sometimes complain of sharp pains or of paræsthesiæ in the form of numbness. Areas of relative analgesia have been recorded in a few instances.

The onset of the disease may be sudden or insidious, and the course may be as short as a few weeks or be prolonged over many years. An early fatal termination is not common, and cases of complete recovery certainly occur. Perhaps the majority of cases run a prolonged course with considerable remissions and relapses, a more or less partial disability for effort of any kind remaining as a permanent feature.

Prognosis.—Owing to the occurrence of marked remissions followed by relapses in the natural history of the disease, to the sudden complication of an apparently benign case by respiratory failure, and to the spontaneous recovery of others, it is difficult to give a prognosis in regard to an individual patient. The condition must always be regarded as a serious one, and only guarded statements can be made in relation to the prospects of recovery.

Diagnosis is often difficult in slight cases, especially in the absence of serious disturbances of the muscles supplied by the cranial nerves. Mere weakness and fatigue, without signs of organic disease, are apt to be regarded as neurasthenic rather than myasthenic in origin. The incidence of the affection on the ocular muscles, the absence of marked myatrophy, and the great variability in the severity of the symptoms suffice to distinguish the disease from ordinary bulbar paralysis. Anomalous forms of peripheral neuritis may present superficial resemblances, but the lack of characteristic sensory, reflex, and electrical changes should determine the diagnosis. Cases of severe ophthalmoplegia with little involvement of other muscles may not be easy to distinguish from the results of cerebral syphilis or of some forms of encephalitis, but the history of onset and the course of the disease will usually set doubts at rest. A myasthenic reaction in response to voluntary effort or to the faradic current often affords help in arriving at a de-

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cision, but must not be regarded by itself as pathognomonic.

Treatment.—The general condition of the patient must be cared for by securing rest, good food, warmth, and freedom from mental disturbance. Little benefit has been derived from massage, or electrical treatment, or the administration of tonics. In view of the possible disturbance of internal secretions, a large number of glandular preparations have been tried, but no consistently good results have been obtained. I have seen symptoms disappear under the administration of thyroid gland by the mouth, and particularly after daily injection of 1 c.c. of pituitrin, but these methods have failed completely in other cases.

Certain symptoms must be met by appropriate measures. When deglutition is difficult the danger of choking must be averted by rectal feeding, the use of the stomach-tube not being permissible on account of the emotional disturbance and consequent exhaustion attendant on its employment. It is advisable to give concentrated food in the early part of the day, when the patient is best able to swallow, and to provide semi-solid nutriment which does not require the effort of mastication. Attacks of dyspnoea may respond to artificial respiration and the administration of oxygen, the tongue being drawn forwards during the process. Patients have occasionally survived several such attacks, and lived in comparative comfort for many years afterwards.

E. FARQUHAR BUZZARD.

MYASTHENIC REACTION (see ELECTRIC REACTIONS).

MYATONIA (see MYOPATHY).

MYATONIA CONGENITA (see AMYOTONIA CONGENITA).

MYCETOMA (see MADURA FOOT).

MYCOSIS FUNGOIDES.—A chronic inflammatory dermatosis with a tendency to form granulomatous tumours which take on malignant characters and usually end fatally.

Etiology.—This is entirely unknown. Mycosis fungoides occurs much more frequently in men than in women, and is a disease of late middle life. It is still a matter of dispute whether the condition is an inflammatory one, due to some unknown organism, or is really a new growth of the sarcomatous type. There is some evidence, however, that it

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may be related to lymphadenoma and the leukemias, but this still remains to be proved.

Pathology.—The microscopic character of the lesions varies with the type and stage of the disease, but the most pronounced feature consists in masses of round cells surrounding the vessels in the dermis. The cells have much the same characters as lymphocytes, but can be distinguished from them by the size of the nucleus and the amount of protoplasm present. The epidermis is only secondarily involved.

Symptomatology.—The most frequent early lesions are patches of erythrodermia—a dry, red, and scaly condition of the skin. These patches occur most frequently on the trunk, and are characterized by their persistence, their failure to respond to treatment, and their intense itching. Sometimes the lesions resemble psoriasis, a simple erythema, or an urticaria. In other cases they are moist and resemble an ordinary eczema. In any case they may be localized to a few patches, or more or less generalized. This is often called the premycotic stage. Later the patches show definite infiltration and become raised above the surrounding skin, or nodules appear in the patches. Finally, tumours develop. These vary considerably in size, but average the size of an orange; they are considerably raised above the surface, and may be either sessile or pedunculated. Ulceration eventually occurs in large areas on any part of the body, producing extreme deformity. The patients then become cachectic and die from wasting, septic absorption, or some intercurrent condition. The lymphatic glands, bone-marrow, lungs, liver, spleen, kidneys, and suprarenals, and in rare cases the brain, have been found to be the seat of similar tumours.

In some cases—the so-called *mycosis fungoides d'emblée*—tumours form without preceding dermatosis, but the final course is the same.

Diagnosis.—In the premycotic stage the diagnosis has to be made from seborrheic dermatitis, psoriasis, parapsoriasis, erythema simplex, urticaria, and eczema. The persistence of the lesions, their resistance to treatment, and their intense itching are the factors on which most reliance must be placed. Histological examination by experts may help. In the later stages the diagnosis is easy, the multiplicity of the tumours and their histological characters separating the cases from *sarcoma*.

Prognosis.—The great majority of cases

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end fatally, though one or two apparently genuine cases of recovery are recorded.

Treatment.—The only treatment known to benefit this condition is X-rays; it is of value in any stage. Tumours disappear rapidly under its influence, the erythematous areas clear up, and itching ceases. The improvement is usually only temporary, recurrence being the rule; but persistent treatment may keep the disease quiescent for years. Drugs such as arsenic, antimony, and chaulmoogra oil have been tried, but with very doubtful results.

A. M. H. GRAY.

MYELITIS.—This designation implies an inflammatory lesion of the spinal cord, though softening due to vascular and other disease is occasionally the main pathological change. The disease may take several forms, but an acute local transverse lesion is the most common.

ACUTE TRANSVERSE MYELITIS.—There can be no doubt that in the majority of cases the lesion is of syphilitic origin; it generally occurs within the first few years after infection, occasionally within the first few months. Exposure, cold, and trauma are less important factors than they were formerly supposed to be. Certain cases develop during the course of or after septic infections or infectious fevers, as measles, scarlet fever, influenza, diphtheria, and malaria, or are due to the same organism as is acute epidemic encephalitis. The pathological lesions vary with the nature of the infection. The syphilitic form is a gummatous meningo-myelitis; the soft membranes are thickened and infiltrated by round cells and exudate, the cellular infiltration extends along the sheaths of the pial vessels into the cord, and this, combined with the vascular occlusions that are liable to occur, produces necrosis and softening of the nervous tissues. In some of the infective cases the meninges are scarcely involved.

Symptoms.—As it is most commonly the dorsal segments of the cord that are affected, the clinical features of this form will be described. The onset is acute. Numbness, tingling, or coldness of the feet and legs may precede the loss of power; difficulty in micturition is occasionally the first symptom. Then the legs become weak, often in an almost apoplectic form manner, and are soon quite powerless; sensation is lost, and retention of urine and faeces develops. When the lesion involves the cervical cord the arms are similarly affected.

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If examined at this stage the limbs are flaccid, and may remain so for weeks. Sensation is lost or disturbed to a level that corresponds to the site of the lesion in the spinal cord, and if the damage to this is severe the tendon-jerks disappear; this is, in fact, the rule. The plantar responses are of the extensor type unless the transverse lesion is complete, when it may be impossible to elicit them, or only flexion of the toes is obtained.

The skin becomes dry, and bedsores are liable to develop on parts subjected to pressure. Not infrequently, and particularly when the meninges are involved, the patient complains of pain or a girdle sensation at the upper margin of the anæsthesia.

After about three weeks or so the tone of the muscles reappears and the limbs become gradually rigid; then reflex spasms, generally of the flexor type, occur, and reflex voiding of the bladder may replace retention. In this stage the deep reflexes are exaggerated, and there may be clonus. The amount of power that returns naturally depends on the severity of the lesion; as a rule, the patient regains fair strength in his legs, though the paralysis was at first complete, but the range of movement is usually limited by the coexisting rigidity. Gait, when possible, is consequently feeble, unsteady, and spastic, the patient is unable to raise his feet properly from the ground and stumbles over every obstacle. The arms, when affected, recover less satisfactorily. A fair degree of sphincter control is usually attained, but micturition is frequently precipitate.

ASCENDING AND DISSEMINATED MYELITIS.—These forms may be considered together, as in ascending myelitis the lesions are often disseminated rather than continuous. The onset resembles that of acute transverse myelitis; when first seen there may be merely a flaccid palsy of the legs and disturbances of sensation to the groins; next day the motor and sensory loss may reach the umbilicus and then gradually ascend the trunk till the arms are involved. As the intercostal muscles are then paralysed too, a fatal termination from respiratory paralysis is likely, but extraordinary and unexpected recoveries take place in this form. Sphincter and trophic disturbances similar to those of transverse myelitis occur.

Many cases prove fatal, but the ultimate prognosis in those that live is not bad, as the

injury to the cord, though extensive, is often not very severe.

CHRONIC MYELITIS.—In the sense given here to the term myelitis, no chronic form occurs. Inflammatory lesions of the cord have always a rapid course. This diagnosis has been unquestionably much misused in the past; the symptoms to which it has been applied have been due to compression of the cord, to the effects of chronic constricting meningeal disease, or to disseminated sclerosis (see COMPRESSION PARAPLEGIA).

Diagnosis of myelitis.—If we restrict the term to acutely developing spinal lesions, there is little danger of confusing myelitis with *compression of the cord*, except perhaps by an abscess in connexion with vertebral caries. *Vascular lesions* the result of arterial embolism, thrombosis, or hæmorrhage are rare and distinguished by the sudden development of a condition that remains stationary or improves. The greatest danger is of confusion with *disseminated sclerosis*, which occasionally produces symptoms of an acute transverse lesion of the cord; but there are usually other signs, as nystagmus, diplopia, or tremor, which make the diagnosis easy. Lumbar puncture is a valuable aid, as the fluid in myelitis invariably contains an excess of cells and albumin.

Treatment should be directed to both the disease and its symptoms. Unfortunately, little can be done in infective myelitis except to control any possible source of infection, but the syphilitic form requires prompt and vigorous antisyphilitic treatment. The life and the prospects of the patient often depend on management and nursing in the early stages. The greatest dangers are cystitis with secondary pyelitis and bedsores. At first regular catheterization is required, and must be conducted with rigid asepsis. If, as often occurs, a cystitis develops, suprapubic drainage should be considered. Bedsores can generally be avoided by the use of a water or air bed, frequent change of the patient from side to side, and systematic treatment of the skin. Massage should be commenced early; electricity is rarely of use, and is undesirable, as it tends to excite rigidity and spasms. The danger of contractures as the tone of the muscles returns must be borne in mind; they can best be prevented by frequent passive movement and change of position of the limbs.

GORDON HOLMES.

MYELOMATA

MYIASIS

MYELOMATA.—Benign growths of the marrow-tissue of bones.

Pathology.—*Macroscopically*, a myeloma is a deep-red, very vascular tumour, having a small amount of fibrous stroma with spicules of new bone scattered through it. As it grows it causes absorption of the inner aspect of the compact bone, but at the same time new bone is deposited externally, so that apparent expansion results. In children the tumour usually undergoes cystic degeneration and gives rise to the commonest form of bone-cyst, containing clear brownish fluid, with a thin layer of solid myeloma forming its wall. In adults the tumour occurs before the age of 40, and is solid, sometimes with small cysts distributed through it.

Microscopically, there is a fibrillated stroma containing oval or spindle cells, with which are numerous giant cells.

Symptomatology.—In order of frequency the bones that are usually affected are the upper end of the tibia, lower end of femur, upper end of humerus, lower end of radius, and the alveoli of the jaws. There is a painless, slowly progressive enlargement of the bone, which later gives "egg-shell" crackling. In the cystic form in children spontaneous fracture is often the first sign; this particularly occurs in the upper end of the humerus. The bone enclosing the tumour may be destroyed, and the tumour then grows more rapidly among the soft parts, and may form a large mass, over which the skin is hot and shows the development of large veins.

Diagnosis.—The absence of pain and the slow progress help to distinguish myeloma from *endosteal sarcoma*. X-rays show thinning and expansion of compact bone and destruction of cancellous bone. The outline of the affected area is more clearly defined than in *tuberculous abscess*, and there is no zone of sclerosis as in *Brodie's abscess*. (PLATE 44, Fig. 3, Vol. III, facing p. 551.) The removal of a fragment for microscopy is the only sure method of diagnosis from certain sarcomata.

Prognosis.—After removal the tumour does not recur, but occasionally, after existing as an apparently benign tumour for some time, a myeloma shows malignancy by the development of metastases in other bones and organs.

Treatment.—The compact bone covering one aspect of the tumour is removed and the soft tissue completely scraped away; in the tibia subperiosteal resection is sometimes necessary. It is occasionally necessary, in very

late cases, to amputate. Where spontaneous fracture has occurred, removal of the tumour is followed by good union.

In BENCE-JONES MYELOMATOSIS, multiple tumours having the characters of myelomata occur in vertebrae, ribs, and cranial bones. The disease begins in middle life and lasts for years, causing spontaneous fractures and great distortion. There is great pain, with intermittent pyrexia and profound anæmia; albumose can be detected in the urine. The disease terminates by sarcomatous degeneration with metastases in lymph-glands and organs. Treatment must be directed to the relief of pain, and the risk of fractures from slight injuries must be avoided.

C. W. GORDON BRYAN.

MYIASIS.—The presence of dipterous larvæ in the living body, and the disorders caused thereby. Such disorders may be divided into four groups:

1. Intestinal.
2. Produced by the deposition of ova in various cavities of the body which open externally.
3. Produced by ova deposited on wounds and ulcers.
4. Larvæ in the subcutaneous tissues.

The larvæ of *Auchmeromyia luteola*, which hide by day in cracks in African dwellings, and at night come out and suck human blood, are not included under the definition.

1. INTESTINAL MYIASIS

This is the form of myiasis most frequently met with in this country; it is still more common abroad. In this condition larvæ of many different flies have been found in Europe and in North America. The commonest are *Fannia canicularis*, the small housefly; *F. scalaris*, the latrine fly; and *Musca domestica*, the common housefly. Quite frequently, more than one species is present in the same case. As a rule, infection is accidental and caused by the ingestion of ova or young larvæ in stale bread, rotten fruit, milk, meat, or other contaminated food. In the case of *Acarus domesticus*, the cheese mite, the larvæ are present in cheese; and in *Eristalis tenax*, the rat-tailed larva of the drone fly, and in *Helophilus pendulus* they are present in dirty water or water-cress.

Fannia scalaris and *Calliphora* probably occasionally lay their eggs on the everted

mucous membrane of the rectum during defæcation; such cases belong to the second group.

Ova and larvæ of most of these flies are uninjured by the gastric juice, and in the favourable conditions present in the intestine—plenty of liquid food and a uniform high temperature—they grow very rapidly. The larvæ of the housefly probably reach their full size in three or four days. Larvæ and pupæ are passed in the fæces, or sometimes larvæ find their way out of the anus by their own movements; more rarely they are vomited. The numbers present may be few, more often they are numerous, and many thousands may be present. The infection is usually a brief one, but cases lasting months or years sometimes occur, and are doubtless due to repeated infections from an undiscovered source. Individuals of any age or station in life are attacked, but people of dirty habits are naturally most exposed to risk.

Symptoms.—Sometimes symptoms are absent; in other cases there are anorexia and discomfort, and in yet others there may be severe attacks of pain with or without diarrhœa. Inflammation with a considerable extent of ulceration may occur in the bowel and cause the passage of blood and mucus. Reflex convulsions and other nervous symptoms have been met with. Recovery is usually rapid and complete, but in a few cases patients have become unconscious and died.

Treatment consists in the administration of a purgative such as calomel, repeated if necessary, and rectal lavage. The active peristaltic movements of the intestine are sufficient to remove the unresisting larvæ. Bismuth or bismuth and opium may be employed later if the intestine is inflamed or ulcerated.

2. MYIASIS OF CAVITIES

The nasal cavities, frontal and other accessory sinuses, mouth, external auditory meatus, conjunctiva, the urinary meatus, urethra and bladder, vagina and rectum, have all been the seat of myiasis. The most dangerous fly is the *Chrysomya macellaria* (screw-worm), a native of America, especially of the tropical and subtropical regions. Other cases are due to *Lucilia cæsar* (greenbottle fly), *Calliphora vomitoria* and *C. erythrocephala* (the blow-flies), *Æstrus*, *Sarcophaga carnaria*, and *Wohlfahrtia magnifica*. The first few of these are found in England, the last is very troublesome and dangerous in Russia. Extensive ulcera-

tion and necrosis of soft parts and even of bone may be produced, and death often follows from secondary bacterial infections. The nose, conjunctiva, auditory meatus, urinary meatus, and vagina are only involved when a pre-existing muco-purulent discharge has attracted the fly.

Vaginal infection may lead to the production of vesico-vaginal or of recto-vaginal fistulæ.

Nasal infection has occasionally led to involvement and destruction of the eye in the case of *Calliphora*, *Hypoderma lineata*, and *H. bovis* (warble flies). The larva tracks along the arteries, reaching the interior of the eye by following the arteria centralis retinæ. Conjunctival infection is very common in Russia and Mexico (*mal de ojo*), and may lead to loss of the eye.

Cases are often characterized by pain, œdema, bloodstained offensive discharge, fever, and delirium.

Treatment must be adopted to get rid of the larvæ; extensive scraping away of necrosed tissue may be necessary, or the injection of pure chloroform, 1 or 2 drachms, followed by antiseptic douching.

3. MYIASIS OF WOUNDS AND ULCERS

Neglected wounds and ulcers are infected by species of *Chrysomya*, *Lucilia*, *Calliphora*, and *Sarcophaga*. Very rapid extension of inflammation and necrosis may follow. Treatment must be similar to that adopted for Group 2.

4. SUBCUTANEOUS MYIASIS

Several species of *Gastrophilus* and *Hypoderma* cause creeping eruptions, which may last for months. The young larva leaves a thin red track. It moves at night and produces burning pain, usually advancing about an inch and a half in twenty-four hours. Larvæ of *Hypoderma* may also cause local boils. *Hypoderma bovis* causes this condition quite frequently in the Shetlands, but it is very rare in the rest of the British Isles.

The larva must be extracted with a small knife, or destroyed by injecting novocain.

In the tropics local boils are quite commonly produced by larvæ of species of *Cordylobia*, *Dermatobia*, and *Bengalia*.

All dipterous larvæ are difficult to recognize, most of them being smooth and maggot-like, though those of *Fannia* are covered with long bristles. They should always be referred to

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a competent entomologist to be named, and if possible adult flies should be bred from the larvæ or pupæ obtained in the first three groups of cases. The source of infection can then be traced in many instances, and further danger avoided.

E. A. COCKAYNE.

MYOCARDIAL DEGENERATION, PROGRESSIVE. Etiology.—The exact causation of progressive myocardial degeneration, one of the chief causes of death, is imperfectly understood.

Predisposing factors. *Age* is the most important. Progressive myocardial degeneration is rare before 50, and practically unknown before 40, except in rare forms such as that accompanying atheroma of the pulmonary artery. This age-incidence is related to the onset of deterioration of the arteries, which occurs about the age of 35, and to the "change of life" in men as well as in women.

Sex.—General experience suggests an equal incidence upon men and women.

Heredity plays its part, presumably similar to that played by age. In certain families the arteries wear out too soon, and the cardiac muscle follows suit.

Occupation.—Such obvious points as the predisposing effect of lead must be noted. More important is the effect of head work as compared with hand work. A collective research in Austria before the War indicated that arterio-sclerosis is attributable to worry more than to manual labour; this seems to be equally true of "cardio-sclerosis." It is a disease of towns rather than of the country.

Exciting causes.—These may be mechanical or toxic. The former group, consisting of manual labour and high arterial tension, almost melts away on examination, for it is hard to prove a greater incidence of cardiac degeneration among men engaged in heavy bodily work, while in cases of high arterial tension it is impossible to deny the active influence of toxins on the cardiac muscle.

Lead is an occasional but definite factor. *Alcohol* is sometimes a very definite cause, producing a syndrome so clear-cut as to constitute a disease in itself—the alcoholic heart. The influence of tobacco is uncertain, but cannot be denied. The association of cardio-sclerosis with *gout*, and also with *glycosuria* of the "diabète gras" type, is well known; but whether the cardiac lesion and the metabolic disorder are effect and cause, or collateral effects of some common cause, cannot clearly

be determined. *Obesity* is certainly a cause, partly, at least, by mechanical means. *Prolonged hyperthyroidism* inflicts injuries upon the cardiac muscle which persist after the cause itself has ceased to operate, and possibly other endocrine secretions have a similar influence. The course of "irritable-heart" cases will be watched with interest in this connexion.

As to *infections*, syphilis is considered elsewhere (see HEART, SYPHILITIC DISEASE OF). Influenza and the respiratory catarrhs undoubtedly hasten, even if they do not initiate, progressive myocardial degeneration. Gall-bladder infections and pyorrhea have also been cited.

The chief causal relation of progressive myocardial degeneration, however, is with *arterio-sclerosis*, *high arterial tension*, and *renal disease*; sometimes with all three, sometimes with two, and sometimes with one. What the order of the relationship may be, cannot yet be stated. There are cases in which it seems certain that nephritis has caused high tension, and high tension has caused myocardial breakdown either directly or through the arterial disease to which it leads. But even here it is possible that the same toxic factor that acted as a pressor has also exerted a directly injurious influence on the myocardium. Perhaps the best statement of the facts as yet available is that high arterial tension, with or without manifest renal disease, causes ventricular hypertrophy and ultimate degeneration; that certain toxic states appear to damage myocardium and arteries simultaneously; and that the arterial decay which marks old age is accompanied by deterioration of the cardiac muscle also.

Pathology. Progressive myocardial degeneration, arising as it does from a multiplicity of factors, cannot be described as a pathological entity. The ventricles, either or both, may be hypertrophied, with or without dilatation. The aorta and coronary arteries are often atheromatous, and the process of calcification often extends to the aortic cusp of the mitral valve and the demilunes of the aortic valve. The aortic ring may be stretched and the valve leaking. To the naked eye the cardiac muscle appears little altered, though in true senility there is sometimes a brown atrophy. Fatty streaks may be visible beneath the endocardium of the ventricles. Microscopically, such methods as are generally used show but little in comparison with the profound physiological disturbances that are

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known to be present: fatty changes within the cells and fibrous formation between them are crude and uncertain criteria.

The various conditions by which the myocardium is brought to downfall operate by disturbing the balance between anabolism and catabolism. Of all the forms of breakdown that may occur, the most serious is failure of left ventricular contractility. Death is due to this even when its apparent cause is some other physiological aberration, such as paroxysmal tachycardia, auricular fibrillation, or heart-block. These—themselves due to failure of anabolism—kill by adding to the burdens of a ventricle which is already expending contractile substance faster than it can make it.

Symptomatology.—The three chief symptoms are dyspnoea, pain, and oedema. The degree of exertion needed to cause dyspnoea is, in the early stages, the soundest gauge of the progress of the disease. Another important and threatening type is the dyspnoea which comes on in apparently spontaneous attacks, usually at night. Pain is another index, hardly less delicate if it is present at all, of the downward progress of the case. Fortunately, it is by no means general. Every degree is experienced, from the mild oppression or constriction over the sternum that assails many elderly men if they try to hurry on a cold day, to the terrible and unspeakable agony of coronary thrombosis. One must recollect, however, that people vary widely in their capacity for feeling pain, as also for bearing it. (Edema of the feet is occasionally the first symptom noted, but pain and shortness of breath are much more often so. If the patient is in bed, there is usually some oedema over the lower lumbar spine. Cough is due to pulmonary complications, and bloody sputum indicates gross congestion, infarction, or apoplexy of the lung.)

Among other symptoms, muscular weakness, giddiness, and fainting fits are prominent; they possibly are due as much to changes in the peripheral arteries as to the cardiac decay. Other cerebral symptoms, such as headache, forgetfulness, and the evidences of retinal and intracranial hæmorrhage and thrombosis, are obviously arterial in origin; except for such headaches as are uræmic. The cardio-renal syndrome is made up of three parts, cardiac, arterial, and renal; any of the three may predominate and may be responsible for the final stage.

Physical signs.—The points to notice in examination of the pulse are the condition of

the artery—the brachial is the best to examine—the blood-pressure, the rate, and the rhythm. Arterial thickening and high tension are by no means coincident. The most beaded and deformed arteries are often under no great tension, and the highest pressures frequently coexist with good arteries. It is roughly true that the worse the heart the faster the pulse, but allowance must be made for the influence on rate which may be exercised by specific disturbances of rhythm.

By far the commonest and least important type of arrhythmia is the extrasystole. Total arrhythmia and the alternating pulse are less often encountered, but as they indicate auricular and ventricular failure respectively, they are highly significant. Paroxysmal tachycardia and the various grades of heart-block are relatively infrequent features.

The physical signs are multiform and difficult to classify. Often there are none except weakening of the sounds, especially of the first, at the apex. This is ominous, as is also the discovery of a gallop rhythm over the apex or the left costal border. On the other hand, pronounced evidences of ventricular hypertrophy and dilatation are often found in elderly persons who have no cardiac symptoms at all.

Added to these evidences of myocardial degeneration may be signs indicative of altered valvular function, the commonest being the systolic apical bruit which proves mitral leakage due to stretching of the auriculo-ventricular ring. A similar bruit, of similar origin, is frequently to be heard over the tricuspid area. At the aortic area there may be signs of valvular calcification, particularly a somewhat loud systolic murmur carried into the greater arteries. If this is accompanied by a regurgitant murmur, one must entertain the thought of syphilis as a causal factor, and indeed, in no case of progressive myocardial degeneration can one venture to forget this possibility, or to omit to look for evidences of aneurysm.

The only important sign of pericardial disease is friction, an occasional but sinister feature if it follows a severe anginal attack, because it betrays the presence of gross coronary obstruction.

In the lungs, one must watch for signs of bronchitis, basal congestion, and hydrothorax; in the abdomen, for enlargement of the liver and ascites. The discs should be examined for hæmorrhages, retinitis, and arterio-sclerosis. The daily output of urine ought to be estimated,

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casts searched for, and the functional activity of the kidneys tested.

Diagnosis.—Whether progressive myocardial degeneration is present, is settled by a due consideration of symptoms as well as of physical signs, but it is not always possible to determine its nature. The disease is usually written down as "senile" if the patient is 60 years old or near it, and if there is no evidence of high arterial tension or of alcoholism, syphilis, or other obvious toxic cause. Lastly, no diagnosis of cardiac disease is complete that does not include a statement of the structural and functional changes, so far as these can be ascertained.

Prognosis.—Symptoms are of more prognostic value than signs. Broadly speaking, the gravity of a case is directly proportional to the ease with which symptoms, particularly dyspnoea and pain, are provoked by effort. A little more analysis will show that it is failure of the contractile force of the ventricles that is the critical feature in these cases, and that dyspnoea and pain, and also oedema, are fairly delicate indices of ventricular inefficiency. It is to patients with symptoms but no outstanding physical signs that death is most likely to come suddenly. Auricular failure, paroxysmal tachycardia, coronary thrombosis and heart-block are of importance in that they precipitate more or less rapidly a breakdown of that for which the heart primarily exists—the ventricular force by which blood is thrown out upon its journey to the periphery of the two circuits. Similarly, weak heart-sounds, "bruit de galop," and the alternating pulse are unwelcome phenomena because they are direct evidences of ventricular failure.

Treatment.—The lesions under consideration are progressive, and associated in a majority of cases with the tissue changes indicative of old age. Obviously, they provide no promising field for radical treatment. Except in a few cases it is impossible even to treat the cause (alcohol, syphilis, lead, and so on), and in those few the damage already done is irreparable.

As the cause cannot be got rid of, the most that can be hoped for is retardation of the downward progress of the case. Since myocardial breakdown occurs as a result of catabolism exceeding anabolism, it can be postponed by (1) promoting anabolism, (2) discouraging catabolism. The administration of cane sugar, advocated by Goulston of Exeter, and the use of cardiotonic drugs such as digitalis, camphor, or caffeine, which help

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the cardiac muscle by improving the coronary blood supply, are examples of the first kind of treatment. Under the second heading are ranged all those measures directed to reduction of the cardiac load. Of these the most valuable are plans for the regulation of life in cases of high blood-pressure before it has gone too far. Probably more could be done to prevent untimely death from cardio-sclerosis by teaching a sound philosophy of life to children than by any therapeutic measure; but even in later years some people show themselves willing to be taught. The hustling man of business must learn not to worry, to take regular exercise, and to eat sparingly of everything, meat in particular. Advice as to alcohol and tobacco must be tempered with discretion. The *Karell cure*—a period of rest with a diet of milk—should be reserved for refractory patients of this type. Personally, I do not believe in the *Nauheim treatment*, except as an elaborate method of psychotherapy. The nitrites and other vaso-dilators will do little or nothing to give enduring relief; but to the emergency which manifests itself in cardiac pain—an emergency which probably arises out of a sudden overloading of an inadequate myocardium—the rapid lowering of peripheral resistance afforded by the nitrites is invaluable.

Other symptoms besides pain call for treatment. The attacks of dyspnoea which make the nights so distressing in advanced myocardial disease are best relieved by morphia or omnopon. Often much help is given by allowing the patient to spend the night in an armchair instead of in bed. For auricular breakdown with fibrillation, digitalis or strophanthus should be given in full doses, but a favourable reaction is by no means so confidently to be expected as in the corresponding stage of post-rheumatic heart disease. These same drugs are sometimes of use in cases of ventricular failure with oedema and an alternating pulse. In an earlier stage, when cardiac pain is easily provoked but the patient is not yet laid up, a course of caffeine will often give relief. Gouty, obese, and glycosuric patients will be benefited by appropriate diet.

CAREY COOMBS.

MYOCARDITIS.—This article deals with the general principles that determine the importance of myocarditis rather than with detailed descriptions of the various forms. The myocardium, comprising as it does not only the muscular wall but also the framework, blood-

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vessels, lymphatics, and nerves of the heart, is of vital importance. At one time perhaps rather neglected for the study of valvular disease, in recent years myocarditis has attracted much attention, and new facts have been established, some of them based upon experimental observation, others attained by the use of new instruments. Two important results, both of practical value, have emerged from these studies. The first is the recognition that myocarditis includes two processes—the perivascular and interstitial lesions, and the neuro-muscular changes. If the former be severe and protracted, scars will develop in the cardiac wall; if the latter be considerable, great destruction of the muscle-fibres and consequent weakening of the cardiac energy will eventuate. Syphilis may produce much fibrosis, diphtheria great muscular weakness. The second result is the appreciation of the two systems of muscular fibres in the cardiac wall—(1) the primitive conduction system, disturbance of which upsets the cardiac rhythm, and (2) the propulsive system, damage to which leads to cardiac asystole. Thus, in all cases of myocarditis we have to determine as far as possible whether the interstitial or the neuro-muscular damage is the greater, and whether the conduction or the propulsive system is chiefly damaged.

Many infections injure the myocardium and produce similar symptoms, which, in general terms, are those of failure in the heart's strength. The particular infection stamps its own peculiarities also upon the process. The infection now to be described is the rheumatic.

RHEUMATIC MYOCARDITIS

The rheumatic form is of particular importance, both on account of its frequency and because it illustrates clearly the interstitial and neuro-muscular changes that develop. Nevertheless, a pure rheumatic myocarditis (as distinguished from a carditis) sufficiently severe to prove fatal is a rare event.

Pathology.—The writer has demonstrated that the *interstitial* lesions in this form do not arise by direct extension from the pericardium or endocardium, but by scattered foci in connexion with the terminal radicles of the coronary blood-vessels in the heart-wall. Aschoff and Carey Coombs have called these lesions "submiliary nodules." Coombs gives the following description of their structure: "The submiliary nodules in myocardial rheumatism consist of rounded or fusiform areas formed by

large spindle-shaped cells, lying in the intra-muscular trabeculae of connective tissue." Their average length is estimated at about 400 μ . They frequently arise in connexion with an arteriole, either around it or even in the wall itself, and their eventual fate would appear to be either cloudy swelling or cicatrization. They contain multinucleated cells larger than fibroblasts, which are surrounded by plasma cells, and by leucocytes which are chiefly mononuclear. In Coombs's experience extensive fibrosis of the heart-wall is very exceptional, and he lays more stress upon the *toxic damage to the muscular fibres and nervous elements* as the cause of the cardiac weakness in myocardial rheumatism. In virulent cases much fatty change takes place in the muscular fibres, and experiment supports the view that such changes are the direct result of the rheumatic infection.

It is clear that scattered foci of the character just described may in some cases damage the lines of communication of the conduction system. By the recent methods of cardiac investigation, evidence is accumulating of the occurrence of partial or complete heart-block and of the development of premature contractions in myocarditis. Though these observations indicate myocardial change, they must be studied and weighed still further before they can be assigned their true position in the study of rheumatic myocarditis. It is certain that they may be quite evanescent.

Symptomatology.—The clinical symptoms and signs are of varying severity, and enable us conveniently to recognize four types of the disease:—

1. Acute fatal myocarditis.
2. Acute myocarditis as a part of a severe rheumatic carditis.
3. Acute dilatation of mild degree.
4. Chronic dilatation of mild degree.

1. **Acute fatal myocarditis** is very rare; the signs are those of virulent rheumatic poisoning together with cardiac asystole. The patient, generally a child or an adolescent, appears anxious and livid. Breathing is rapid, and may suggest pneumonia or miliary tuberculosis. The pulse is rapid, low in tension, and almost imperceptible. The condition of the heart is one of extreme dilatation with a wide area of flickering impulse. The cardiac sounds are short and approximate, and there may be a soft systolic murmur in the mitral area. Vomiting and restlessness supervene, the liver enlarges, and slight cedema of the extremities

MYOCARDITIS

and albuminuria occur. The temperature may be subnormal, but is not a characteristic feature. Death is usually sudden.

2. The **subacute form** is more common, and its symptoms play a prominent part in all examples of severe rheumatic carditis. Its presence is suggested by the occurrence of persistent cardiac rapidity, failure in compensation, and irregular fever in the absence of pericarditis, and this suggestion obtains support from such additional signs as orthopnoea, pallor, general weakness, slight persistent oedema, and progressive enlargement of the liver. A point worth emphasizing is that many cases of rheumatic heart disease with myocardial damage are graver than those of frank pericarditis which run a rapid and benign course.

3. **Acute dilatation of mild degree** is the most frequent cardiac event in rheumatism. (See **ENDOCARDITIS**.)

4. The **chronic form** is of more importance than is perhaps generally recognized. In such cases the heart remains large and feeble, and the pulse rapid and easily influenced by slight disturbances for long periods. There are signs of moderate dilatation, often accompanied, in childhood, by marked nervousness. Syncopal attacks and palpitation are not uncommon. These cases are very obstinate, and any overstrain will rapidly undo all the good produced by months of patient waiting. Eventually there may be an excellent recovery, but some of the patients drift into a state of chronic invalidism.

Diagnosis.—The chief difficulties in diagnosis are the exclusion of acute *pericarditis* and of a large *pericardial effusion*. The first is somewhat academic, for the chief danger of acute pericarditis is the attendant myocardial damage. The second is important, for the decision will determine the method of treatment. The diagnostic points in a case of copious pericardial effusion are given under **PERICARDITIS**. The guiding rule that dilatation is frequent and a large effusion uncommon in acute rheumatic carditis is a valuable check to rashness.

Prognosis.—In the virulent cases the prognosis is very bad; in the severe cases of carditis, serious; in the acute mild cases, good; and in the chronic cases, good with some considerable reservation.

Treatment is similar to that of acute endocarditis (see **ENDOCARDITIS, ACUTE**). There is need for even more caution and patience than in valvular affections, for a false step may mean a long delay in future progress.

F. J. POYNTON.

MYOPATHY

MYOCLONUS MULTIPLEX (see **PARA-MYOCLONUS MULTIPLEX**).

MYOPATHY.—A condition in which the skeletal muscles, from congenital defect and subsequent pathological change, undergo atrophy. It is not associated with any lesion of the nervous system, and is characterized by progressive muscular wasting and weakness developing during infancy or childhood. Although, clinically, the disease falls into several well-defined types, mixed and transitional cases occur, and in all its forms the disease is essentially the same. Erb has named the condition *Progressive Muscular Dystrophy*.

Etiology.—The cause is unknown. It probably depends upon a qualitative defect in the embryonic tissue in which the muscle arises. It commonly occurs in several members of a family, or in several generations. Both sexes transmit the disease and are affected by it. The pseudo-hypertrophic form predominates in males, the other forms affect both sexes more or less equally. The former type commonly runs through one family, and is not often seen in association with the other types. Isolated cases of all forms also occur.

Pathology.—There is decrease in the number of muscle-fibres, and some muscles may show a congenital defect. The interstitial tissue of the muscles is greatly increased, and the completely atrophic muscle may consist of little else. As there is also a deposition of fat in the fibrous tissue between the remaining fibres, some muscles may appear enlarged or hypertrophied. Microscopically the fibres show atrophy, diminution in size, vacuolation, increase of nuclei, alterations in form, and occasionally enlargement. The muscular nerves and motor end-plates are intact. Such changes as have in rare instances been recorded in the peripheral nerves and ventral-horn cells are probably secondary to the muscular atrophy.

General symptomatology.—The disease develops during infancy or childhood; that is, before growth is completed. It is slowly progressive, and, although in the pseudo-hypertrophic form certain muscles may become enlarged, the general tendency is finally to widespread atrophy. Both wasted and enlarged muscles are weak. The loss of power appears first in the muscles of the trunk and limb girdles, the distal limb muscles being less affected throughout. Contracture often ensues in the wasted muscles, producing deformities in limbs and spine.

The initial symptoms vary in the different forms according to the muscle groups affected earliest. In many cases the child has never been normally active and nimble, and may have learned to stand and walk unduly late. Attention is often drawn to the condition by a clumsiness in walking; a characteristic waddling gait, a tendency to fall, and great difficulty in regaining the erect posture. The attitudes adopted in these circumstances are highly characteristic. They are best seen in the pseudo-hypertrophic form, of which they are almost pathognomonic. The child rolls over on to his face, and, getting on hands and knees, proceeds to climb to the upright position by working his hands alternately up his legs and thighs until he is erect.

Weakness of the muscles of the shoulder girdle limits the power and range of active movement at this joint, causes winging of the scapulæ, abnormal mobility of the shoulder girdle when the patient is lifted by hands placed in the axillæ, and an abnormal sagging of the shoulders when the arms are hanging by the side.

A marked lordosis, partly from a tilting forwards of the pelvis due to weakness of the hip extensors, and accentuated by the enlargement of the glutei, is seen when the patient stands upright, but disappears when he is sitting down. The shoulders are thrown well back and the scapulæ protrude.

The muscles of respiration, deglutition, and phonation are immune, as are also the tongue, the oculo-motor and sterno-mastoid muscles.

Certain muscles are especially prone to wasting. These are the lower parts of the pectorals and the trapezii, latissimi dorsi, serrati magni, biceps and triceps, and, in the lower extremities, the hamstrings and adductors. When enlargement is present, this also picks out certain muscles with great constancy—the deltoid, infraspinatus, biceps, the glutei, vasti, recti femoris, and the calf muscles. The erector spinæ and masseters may also be enlarged. The enlarged muscles have a rounded, somewhat exaggerated contour, and are very firm to pressure.

On electrical examination, it is found that as the muscle-fibres progressively atrophy and disappear their excitability both to faradic and to galvanic currents gradually diminishes and finally vanishes. The reaction of degeneration is never present at any stage. Fibrillation of the affected muscles is exceedingly rare, and they are neither painful nor tender to pressure.

The sphincters are always intact, and sensation is normal. The tendon-jerks grow feeble and disappear as the muscles concerned in them waste and lose function. Both knee- and ankle-jerks are commonly lost early.

The fibrosis of the muscles leads ultimately to gross deformities, such as talipes equinus, lordosis, and curvature of the spine. These complete the disability of the patient.

Clinical forms. Pseudo-hypertrophic type.

—This usually occurs in males, and is the most distinctive and readily recognizable type. It develops during the first decade of life, and the early symptoms are those necessarily associated with weakness of the pelvic and thigh muscles, viz. lordosis, a clumsy waddling gait, and difficulty in regaining the erect posture when lying down. The characteristic attitudes adopted in these circumstances have been described above. These phenomena and the presence of massive calves first draw attention to the condition in most cases. Several boys in a family may be affected, and thus the disease may be seen in different stages of its evolution in a single family. The distribution of wasting and enlargement of muscles is that already described. The combination of wasting of the lower fibres of the pectoralis major with enlargement of the infraspinatus and the calves is pathognomonic of this form. Associated with the enlargement of the calves is some permanent contracture, which finally develops into marked equinus. Knee- and ankle-jerks are finally lost, and the general progress of the disease is fairly rapid, rendering the child helpless within a few years. With the exception of occasional enlargement of the masseters, the facial muscles remain intact.

Scapulo-humeral type (Erb).—This occurs in both sexes, appearing in late childhood or at adolescence. There is commonly both an hereditary and a familial incidence, and this and the succeeding type may both be found in members of the same family, but seldom in association with the pseudo-hypertrophic form. Weakness of the shoulder girdle muscles is the early characteristic of the type, and gives rise to the disability and deformities described earlier, so that winging of the scapulæ and sagging of the shoulders are prominent early symptoms. Later, weakness of the pelvic and thigh muscles may be superadded, with accompanying defects of movement such as are seen in the pseudo-hypertrophic form. However, the absence of any definite enlargement of muscles and the initial involvement of the

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upper limb readily distinguish the juvenile from the former type.

Facio-scapulo-humeral type (Landouzy-Dejerine).—This closely resembles the scapulo-humeral type, and may be associated with it in an affected family. The characteristic affection of the face muscles may be present from infancy. The muscles chiefly affected are the orbiculares palpebrarum and oris and the zygomatici. The patient has very little mobility of the face, cannot close the lids, blow out the cheeks, whistle, or smile properly. In the infant, sucking may be defective. The lips are thick and everted, and gape so as to give the patient an expression of vacant stupidity. The oculo-motor muscles and those of the tongue, larynx, and pharynx escape. At a later age, sometimes in childhood, at times not until adult life, weakness and wasting of the shoulder girdle muscles appear. The serratus magnus, latissimus dorsi, the lower fibres of the trapezius, and the upper part of the pectoralis major atrophy and disappear. In other cases it may be the lower part of the pectoralis that is affected. The biceps, triceps, and supinator longus are in turn involved. The thigh and pelvic muscles may also be affected, and then the patient presents the peculiarities of attitude and gait found in association with this distribution of the affection in the other forms of myopathy.

Eventually, talipes and spinal curvature may develop. These different muscle groups are not always involved, and the patient may reach adult life with but a slight affection of the facial muscles and no other disability.

Distal type.—This relatively uncommon type differs from those described, in that weakness and wasting first appear in the distal muscles of the limbs, and invade the proximal segments and limb girdle muscles later as the disease progresses. The face also is affected. This type develops in later childhood, and is differentiated from the peroneal type of muscular atrophy by its spread, the affection of the face, the absence of sensory changes, and the occurrence of other types of myopathy in the affected family.

Mixed and transitional types.—In addition to the clear-cut clinical types described, mixed and transitional cases also occur in which we see the characteristics of more than one type; for example, some pseudo-hypertrophy may be found in the deltoid and the spinati in a case of the scapulo-humeral type, while other cases show a widespread muscular enlargement as

their most characteristic feature. As a rule, however, these cases approximate to one or other type.

Prognosis.—This varies in the different types and in individual cases both as to progress of the disease and as to duration of life. In general, it may be said that the earlier the disease develops, the graver the prognosis as to both disability and life. The usual termination of the condition is from some pulmonary complication in the helpless and bedridden patient.

In the pseudo-hypertrophic type the prognosis is grave. Within five or six years the child is helpless, and does not usually attain adult life. In the facio-scapulo-humeral and scapulo-humeral types the progress is more variable. In those cases in which weakness of the shoulder and pelvic girdle muscles appears early, progress is likely to be more rapid than when the affection is confined to the face till adult life. Contractures and deformities are always more extreme in the pseudo-hypertrophic than in the other forms. Recovery never occurs.

Treatment.—The course of myopathy is not modified by treatment, and when the probability that the disease is developmental and congenital in origin is considered, this is scarcely matter for surprise. Treatment, therefore, has to be purely palliative and symptomatic. From what has been said of the changes in the electrical excitability of the muscles, little good can be anticipated from electrical treatment. In fact, the stimulation of the stronger of two antagonistic groups of muscles may even accelerate the formation of contracture in that group and so aggravate the tendency to deformity. Nevertheless, Erb stated that he had seen arrest follow electrical treatment, and a mild continuous current just sufficient in strength to cause appreciable contraction, if used with these possibilities in mind, may provide a form of gentle exercise of the muscles. Voluntary exercise and passive movements are, however, more effective; they prevent contractures and delay disability. The patient should not be confined to bed, if it can be avoided; carefully graduated active exercises, according to his ability, may definitely retard the progress of the disease.

Drugs and glandular extracts are unavailable. It is important that the general health of the patient should be maintained at as high a level as possible. F. M. R. WALSH.

MYOPIA (see REFRACTION AND ACCOMMODATION, ERRORS OF).

MYOSITIS (inflammation of voluntary muscles).—Inflammation may affect (a) the fibrous parts of muscles, their sheaths, septa, aponeuroses, and tendons, or (b) the muscle-bundles themselves. The former variety is considered under MYALGIA, and is comparatively common; the latter as a separate disease is very rare. An *infectious or suppurative* form has been described, more particularly in Japan, which begins suddenly with high fever and severe toxæmia; local indurations are found in one or several muscle-bellies; these subsequently suppurate, and the resulting abscesses call for free evacuation and drainage. There is considerable danger of pyæmia. The organisms most commonly found in the pus have been staphylococci.

In *dermato-myositis* not only muscle but skin and subcutaneous tissues are affected. The disease is of unknown etiology, and may occur either in adults or in children. It may be acute or subacute, while remissions are common. Usually beginning acutely, there is fever, accompanied by swelling and redness of the skin, and perhaps localized œdema, in several areas. With the subsidence of the dermatitis the underlying muscles are found to be tender and unyielding. After persisting for some months the inflammatory changes may disappear completely and recovery result. More often recrudescences occur, the skin and muscles remaining thereafter indurated and thickened. The disease is often fatal, though death may be postponed, the patient becoming emaciated, with contracted limbs, and eventually succumbing to an intercurrent disease. The presence of paræsthesia and of anæsthesia in some cases has suggested the name *neuro-myositis*.

In a case of a child, described by F. E. Batten, pronounced atrophy and parenchymatous degeneration of the muscle-bundles were found, especially in their more superficial parts and near the intermuscular septa. The deeper fibres were comparatively normal. Between the muscle-fibres were lymphorrhages, and there was perivascular lymphocytosis. The walls of the vessels were thickened, amounting in places to obliteration of the lumen, a change which affected the vessels not only of the skin and muscles but also of the viscera. The skin was atrophied and the subcutaneous fat replaced by fibrous tissue.

Polymyositis hæmorrhagica differs only from dermato-myositis in the presence of hæmorrhages, which may occur into or between the muscles, into the skin, or from mucous membranes.

FREDERICK LANGMEAD.

MYOSITIS FIBROSA.—A rare disease in which the voluntary muscles become infiltrated with fibrous tissue. Without pain or discomfort, indurated areas appear in the muscles and gradually spread until most of the palpable muscles become hard and almost tendinous in consistence. Ossification does not occur in them. The disease begins in early life, and in most of the cases described has affected first the legs, then the muscles of the neck and back and arms, and finally those of the thorax and abdomen. Though usually progressive, it may become stationary, and in a few cases gradual recovery has taken place. In a case of mine the condition was obvious within a few weeks of birth and was apparently congenital in origin. Hardening of skin and subcutaneous tissue was also present. As the result of shortening of the muscles and fixation of the joints, gross deformities result, the patient becoming rigid with spine and limbs flexed. In a case recorded by F. E. Batten the great toes were proportionately short, and in my own case the first toes were abnormally large and long. Batten thus described the pathological findings in his case: "The muscles were firm and hard, and when cut across consisted of a tissue which grated under the knife and appeared hard and white on section. In less affected parts the muscle was recognizable as reddish-yellow points on a white ground. Microscopically a mass of interstitial tissue was seen between the muscle-fibres, which in part showed granular degeneration and in part were atrophied. In the more affected portions the muscles were composed entirely of tendinous tissue."

The **diagnosis** is from myositis ossificans and dermato-myositis. From the former it differs in the absence of bony change in the muscles, as proved by X-rays. From dermato-myositis it is distinguished by the absence of early dermatitis and constitutional symptoms. Only when the skin and subcutaneous tissues are involved is the distinction difficult.

The only **treatment** likely to benefit is massage, hot-air baths, passive movements, and electricity. If the disease becomes stationary the deformity may be lessened by suitable orthopædic measures. FREDERICK LANGMEAD.

MYOSITIS OSSIFICANS PROGRESSIVA.

Ossification beginning in the substance of voluntary muscles, and afterwards involving fasciæ, tendons, ligaments, aponeuroses, and perhaps becoming attached to bone. The disease is rare, and its etiology unknown. It begins in early life, and has been described in a child of five months (Garrod). Its onset is characterized by swellings, generally situated in the back and neck, but sometimes in the limbs, and of two kinds. In the first these are circumscribed, rounded protrusions, firm but resilient, attached to deeper structures but not to the skin; the second are less defined and do not project; they somewhat resemble areas of deep-seated œdema but do not pit; over them the skin cannot be moved. The rounded projecting bosses have been seen to divide (Garrod). Some of the swellings disappear completely, but, sooner or later, swellings persist and, perhaps after several months, calcification occurs in them and true bone is produced. The bone appears in the form of discrete small patches, recognizable by X-rays. Later, by enlargement and coalescence, irregular bony plate-like masses of considerable size develop.

Though the muscles of the neck and back are those most often affected, any muscle may be involved. The disease slowly increases, progressively causing crippling by loss of mobility of joints or of the contractility of muscles. The scapulæ become fixed to the thorax, the spine rigid, and the limbs immobile. Ultimately the patient may become statuesque, unable to sit up or turn over, to move the head, to flex the elbows or knees, or to assume the erect position, though he retains this position if placed in it. Ossification of the masseters and temporal muscles may prevent eating, so that the patient, unable to help himself, may need to be given fluids from a feeder or spoon. Occasionally the bony plates may be fractured by an injury, or both the bone and muscles of a limb may be broken, as happened to a patient under my observation. Death takes place from intercurrent disease.

A curious but usual association is congenital shortness of the great toes, due either to suppression of the proximal phalanx or union of stunted first and second phalanges. Less often there is a similar malformation of the thumbs.

Diagnosis.—The disease must be distinguished from the myositis ossificans which results from injury and occurs in muscles in

proximity to a damaged bone; this also may not be attached to bone. *Osteomata* and *bony spurs* ought not to be confused with it, for they arise not within the bellies of the muscles but from the bone in the neighbourhood of the epiphyseal lines and of the tendinous insertions respectively. In *calcinosis* (q.v.) the calcification is more superficial, and is evidently situated in the subcutaneous tissues.

No treatment has been found to influence the disease.

FREDERICK LANGMEAD.

MYOTONIA ATROPHICA.—This rare disease is characterized by muscular atrophy of peculiar distribution associated with myotonia, or tonic spasm and delayed relaxation of certain muscles on voluntary contraction. The cause is unknown, but, as in the other primary muscular diseases, it probably depends upon a developmental defect. It may occur in several members of a family, but its hereditary nature is less certain. Males are most commonly affected. Of its morbid anatomy little is known, but in this respect it is allied to the myopathies.

Symptomatology.—The onset is during early adult life. The myotonia develops before atrophy, and resembles in character that found in Thomsen's disease (see MYOTONIA CONGENITA). It is most obvious in the face and tongue muscles. Affected next in degree are the distal muscles of the limbs, particularly the flexors of wrist and fingers. Relaxation of grasp may take from five to fifteen seconds. Percussion of the tongue produces a characteristic prolonged local contraction and wrinkling of the mucosa. The lower limbs are less severely affected; in them myotonia may be found in the calf muscles only.

The atrophy has a characteristic distribution. The face muscles and the sterno-mastoids are much wasted, and there is always a pronounced myopathic facies, such as has been described in the Landouzy-Dejerine type of myopathy (see MYOPATHY). The vasti, dorsi-flexors of the feet, and the forearm muscles also waste. The intrinsic hand muscles are less frequently affected. The tendon reflexes are usually absent. Sensation is normal, and the sphincters are intact.

In some families a peculiar form of cataract is found associated with this disease, while mental disorders, as psychasthenia and melancholia, have also been recorded in connexion with it.

MYOTONIA CONGENITA

The progress of myotonia atrophica is very slow, and in some cases is scarcely appreciable; beyond limiting the patient's activity, the disease has but little effect. There is no treatment beyond the maintenance of the general health. Massage has no permanent effect.

F. M. R. WALSHE.

MYOTONIA CONGENITA (Thomsen's Disease).—A familial muscular disease of obscure origin and rare occurrence, characterized by a peculiar tonic spasm and retarded relaxation of the muscles on voluntary effort after a period of rest. This myotonia, as it is called, is transient, and does not recur during a period of activity. The cause is unknown. The disease is hereditary, and may occur in several members of a family; both sexes are affected, males most frequently. The onset of symptoms is during early childhood.

Pathology.—The disease is probably due to a congenital developmental defect of the muscle-fibres, which are abnormally large. Microscopically they are found greatly increased in diameter, their striation is poorly marked, and the sarcolemmal nuclei are increased in number.

Symptomatology.—The symptoms usually appear during childhood, and consist in a peculiar slowness and clumsiness of movement, which is always worse after rest, particularly on rising from bed in the morning. When walking, movements of the arms and hands are particularly difficult on account of the fixation of the patient for a few moments in an attitude from which he cannot escape until slow relaxation is complete. During the course of movement this wears off, leaving the patient's activities fairly normal, till he rests again for a short while. Muscular strength is good, though not in proportion to the bulk of the muscles, which may show a widespread enlargement of a "herculean" character. The tonic rigidity described is characteristic of the disease. It is widespread, affecting most severely the lower limbs. The muscles of the face and of the eye, and those of deglutition, phonation, and respiration are unaffected. The sphincters are intact. The myotonia is best brought out by forceful movement, such as a firm hand-grasp. Percussion of the affected muscles gives a slow sustained contraction, while to faradism and galvanism the muscle responds by a maintained contraction lasting several seconds.

The progress of the disease is extremely slow,

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and apparently arrest may occur during adult life. The condition is at times associated with mental disorder. Treatment does not influence its course. Systematic exercise and maintenance of the general health are all that can be attempted.

F. M. R. WALSHE.

MYXEDEMA.—A chronic malady due to loss of function of the thyroid gland by disease or by removal.

Etiology. Exciting cause.—In a few cases the disease has developed in consequence of syphilis, actinomycosis, or suppuration of the thyroid gland, or it has followed recovery from Graves's disease. In the majority of cases no direct exciting cause can be found.

Predisposing causes. Age.—Myxœdema may develop at any age, but the tendency gradually increases up to the age of 45 and then declines. **Sex.**—The malady occurs about seven times as frequently in women as in men. **Heredit.**—Two or more cases may occur in a family, but the disease has rarely appeared in two generations. **Locality.**—It is chiefly a disease of cold and temperate climates, and affects white rather than coloured races.

Pathology.—Myxœdema is due to loss of function of the thyroid gland, as is proved by the following observations: (1) In man the gland is found to be atrophied in advanced cases. (2) The symptoms of myxœdema appear in man and in some lower animals when the gland is removed by operation. (3) The symptoms of the disease disappear when an adequate amount of thyroidal hormone is supplied from an external source.

In the early stages the walls of the alveoli of the gland are infiltrated with small round cells; in the later stages the number of alveoli gradually diminishes, and they are replaced by fat and fibrous tissue till the whole gland is completely atrophied and only weighs from 3-5 grm. As the normal stimulus to metabolism which is given by the thyroidal hormone is lacking, the basal metabolic rate may fall to -30 per cent. or -40 per cent. In those who have died in an advanced stage of myxœdema the skin is thickened, and in the connective tissue of the corium the normal trabeculae are replaced by bundles of nucleated fibrillae. There may be endarteritis of the cutaneous vessels, with atrophy of the sweat- and sebaceous glands and of the hair-follicles. In advanced cases fatty and fibroid degeneration of the cardiac muscle may be found. Chronic interstitial nephritis

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may be present and may be the actual cause of death.

Symptomatology.—The onset of the symptoms of primary myxedema is very gradual, as the atrophy of the thyroid gland comes on slowly. When the disease is secondary to removal by operation the onset may be rapid if the thyroidectomy has been total. In the early stages of the malady an increasing disinclination for effort and sensitiveness to cold are the chief subjective symptoms. The face gradually changes in appearance owing to the subcutaneous swelling (PLATE 21), which is most easily noticed in the eyelids, lips, and cheeks, the cheeks being also slightly flushed. The skin often acquires a yellowish tint and becomes dry, while the hair begins to come out. Even in this early stage slight visual and auditory hallucinations may occur. The temperature may be normal or only slightly subnormal, but as the disease advances it falls one or two degrees below normal, and it may be as low as 95° or even 93° F. The various other symptoms gradually become more pronounced, and may now be considered in detail.

Cutaneous system.—The skin and subcutaneous tissues are so profoundly altered that the entire aspect of the patient is changed. The subcutaneous tissues are swollen by the solid oedema, which does not pit on pressure and may add several stones to the weight. The swelling extends all over the body, but is most apparent in the eyelids, cheeks, lips, chin, and on the dorsal surface of the hands and feet. Owing to the swelling of the upper eyelids there is an instinctive contraction of the occipito-frontalis muscle with transverse wrinkling of the forehead. The hands and feet look broad and clumsy, and the movement of the joints is impeded by the swelling. The skin is dry, and there is often a fine desquamation. Perspiration ceases, the hair is fine in texture and sparse, while the eyebrows and eyelashes are scanty.

Mental symptoms.—Slowness of apprehension and delay in response are marked characteristics. The temper is normally placid, and the memory for recent events is impaired. Patients are conscious of their altered appearance, and this, combined with the general hebetude and sensitiveness to cold, causes a preference for a secluded indoor life. In a few untreated cases acute or chronic mania, melancholia, or dementia may develop.

Special senses.—Sight is defective in about half of the cases, though in many this is not

due to the thyroidal insufficiency. Defective hearing is equally frequent and may affect one or both ears. The senses of taste and smell are frequently diminished. Sexual feeling is generally lessened, and sterility usually results in women but not necessarily in men. Sensations of cold are commonly felt and tactile sense is diminished. Headaches and neuralgic pains in various situations may take place.

Circulatory system.—In the early stages of the malady no special symptoms of cardiovascular disease are detectable. In the later stages, when degenerative changes in the myocardium ensue, dyspnoea and palpitation on exertion often occur, and there may be attacks of angina pectoris or of syncope, either of which may prove to be fatal. The cardiac impulse is feeble, and the heart-sounds are distant, but no murmur is heard unless there is an antecedent valvular lesion. The pulse-rate is slow but regular, and the blood-pressure is raised if arterio-sclerosis or renal disease is present.

Blood.—Some degree of anæmia is common, the number of red corpuscles being reduced to between three and four million to the c.mm., and the hæmoglobin to 60 or 70 per cent., or even to less than 50 per cent., of the normal amount.

Digestive system.—The appetite is generally poor, and constipation is common. The lips, tongue, and gums are swollen, and the teeth are frequently carious. There is often a thick mucous discharge from the mouth during sleep.

Genito-urinary system.—The skin covering the external organs of generation is dry and swollen and the pubic hair is scanty. Owing to the age at which the disease develops, pregnancy is very rare. In early cases in women there may be menorrhagia, though in those more advanced amenorrhœa is usual. In an early stage the urine is normal, but later may be pale, of low specific gravity, and contain less than the normal amount of urea. In about 20 per cent. of cases there is albuminuria.

Diagnosis.—When the disease is fully developed the diagnosis can readily be made from the facial appearance of the patient. In the early stages the dryness and slight swelling of the skin, loss of hair, lethargy, sensations of chilliness, and slight hallucinations are important signs. The swelling may be distinguished from that of ordinary obesity by



PLATE 21.—MYXŒDEMA.

(Dr. Longmead's case.)

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the translucency of the eyelids and the presence of some of the above early symptoms. The absence of pitting on pressure of the skin and the character of the urine will distinguish the condition from *chronic nephritis*, for which it has sometimes been mistaken. In *adiposis dolorosa* the subcutaneous swelling is due to irregular deposits of fat, which are painful and tender on pressure, and the face, hands, and feet are not affected. In *acromegaly* the change in appearance is due to definite enlargement of the bony and other structures and not to subcutaneous swelling. In cases of doubt a course of thyroid treatment may be given for a month. If the symptoms are due to hypothyroidism, definite improvement will follow.

Prognosis.—In untreated myxœdema the disease tends to get progressively worse. The change may be very gradual, as some patients have continued an invalid existence for more than twenty years. Since the introduction of treatment by thyroid extract the outlook has entirely changed. Under the influence of continuous treatment a healthy life may be enjoyed for many years.

Treatment.—It is important to explain to the patient that the principle of the treatment of myxœdema is simply to make good a physio-

logical deficiency of thyroïdal hormone, the supply of which must be maintained *permanently*. In an early case this can be accomplished most readily by giving 5 min. of freshly prepared *liquor thyroidei* (B.P. 1898), or 2 or 3 gr. of dried thyroid in a tablet, each night at bedtime. In more advanced cases the dose should gradually be increased to double these amounts. In advanced cases, especially if there are any symptoms of myocardial degeneration, even smaller doses should be given at first, and gradually increased to the full amount, the patient being kept at rest meanwhile and only allowed to resume exercise by degrees. Any marked increase in the frequency of the pulse, rapid loss of weight, or aching pains are indications that the dose is too large. Under the influence of the treatment the swelling disappears, the skin becomes moist, the hair grows again, basal metabolism returns to the normal level, and the patient recovers. The symptoms do not return so long as the treatment is continued. My first patient, who was in an advanced stage of the disease when the treatment was commenced in April, 1891, lived for twenty-eight years in good health, and died in May, 1919, of heart failure at the age of 74.

GEORGE R. MURRAY.

NÆVO-CARCINOMA (*see SKIN, MALIGNANT GROWTHS OF*).

NÆVUS (*syn.* Angioma).—Local hyperplasia and new formation of vascular and to a less extent of other tissues.

Nævi are present at birth, or make their appearance soon after. During the first few months of life they may enlarge rapidly. They seldom disappear spontaneously, and many persist through life unchanged. They vary in size from a pin's head to an orange, and may swell up or pulsate when the patient coughs or cries.

Clinical groups.—Nævi on the surface of the body are divided into the following clinical groups: (1) *Cutaneous* nævi (angioma simplex, capillary angioma, port-wine mark); (2) *subcutaneous* nævi (cavernous angioma); (3) *mixed cutaneous and subcutaneous* nævi; (4) *spider* nævi. (For other cutaneous growths to which the name of nævus has been attached, *see*

MOLES.) The *cutaneous* nævi form bluish-red-stained areas of skin, not raised above the general level of the surface. *Subcutaneous* nævi present themselves as flat-topped elevations of soft consistency. They are compressible, and swell when the patient cries or vigorously exerts himself. The skin over them is unchanged, the nævoid tissue showing through with a somewhat bluish hue. Much more common than the last two varieties is their combination, the *mixed* nævus. Here there is the compressible elevation, but it is covered by an area of bright-red skin. The subcutaneous element is more extensive than the cutaneous. Fatty tissue may be present among the nævoid elements (*nævo-lipoma*).

Occasionally an inflammatory process takes place in the nævus, the result of which may be that the blood spaces become thrombosed and obliterated, but more often ulceration occurs and hæmorrhage supervenes.

Treatment.—Excision may be contraindicated by æsthetic considerations, as occasionally when the growths are situated on the face, or by anatomical reasons, in cases in which removal may be followed by a deformity, as when the eyelids are involved. In the face the linear scar of excision may be less disfiguring than the scar left by other methods of treatment or the original mole. The alternatives to excision are freezing with solid carbon dioxide or liquid air, destruction by radium emanations or by electrolysis. For *cutaneous* nævi, freezing with liquid air or solid carbon dioxide is the procedure of choice. The application of radium is effective, but should be done only by the expert. The results are at present uncertain, and the scars no less noticeable than those left after the other-mentioned methods. The *subcutaneous* nævus is treated by (a) excision, (b) puncture with the galvanocautery, (c) electrolysis, or (d) the

there is very little bleeding if the incisions lie outside the boundaries of the tumour.

Puncture by the galvanocautery is useful in cases in which, as so often happens, the nævus is partly without and partly within the mouth cavity; in such cases excision may be neither practicable nor advisable, especially when the nævus involves the whole thickness of the lip. It is also suitable for the larger mixed nævi. A very fine cautery-point at a dull-red heat is employed; the punctures are made about $\frac{1}{8}$ in. apart. The first punctures should be made around the margin of the nævus. This not only stops peripheral growth of the tumour, but may also cut off the blood supply of the central portions. A boric-acid ointment dressing is applied.

Electrolysis is useful for nævi of limited size only. A general anæsthetic is necessary. In the unipolar method a large flat indifferent electrode is attached to the positive pole and applied somewhere to the patient's skin. The needle attached to the negative pole may be made of steel or zinc. A current of 5–10 ma. is passed until bubbles appear. The current should be increased or decreased in intensity gradually, to avoid shock. When the bipolar method is employed, needles are attached to both poles of the battery and inserted into the nævoid tissue. They should be made of an alloy of platinum and iridium. A steel needle should never be attached to the anode, as it will stain the skin. Should the anodic needle become adherent, a short reversal of the current will free it. The periphery of the nævus should be attacked first. After electrolysis a boric-acid dressing should be applied.

The **injection of boiling water** is a simple, effective, and safe method of treating cavernous angiomas, i.e. the majority of subcutaneous and mixed nævi. An anæsthetic is necessary. A syringe with an asbestos plunger is better than an all-glass or glass-and-metal syringe. The vessel containing the actively boiling water should be at the hand of the operator. As in other methods, the margin of the nævus should be treated first. The amount of water to be injected varies with the size of the tumour, but as much as two or three ounces may be used. Injection is stopped when the tumour becomes hard and paler in colour. A great deal of swelling and œdema occurs in the neighbourhood of the nævus, but it is of no moment and soon subsides.

The technique of the application of **liquid**

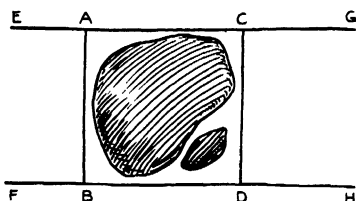


Fig. 51.—To show appropriate incisions for excision of a nævus.

injection of boiling water. *Mixed* nævi are treated by the same methods.

Excision is the quickest and most satisfactory procedure, except when contraindicated by the considerations mentioned, or when a cavernous angioma of the scalp overlies one of the fontanelles, in which position it may communicate with the superior longitudinal sinus. The best incisions are two semilunar cuts enclosing the tumour; their suture leaves a single linear scar. On exposed surfaces of the body, such as the face and neck, make the incision, if possible, lie in a natural crease. To prevent subsequent stretching of the scar the subcutaneous tissue should be sutured with fine catgut. For the skin a subcuticular stitch is used. If the tumour is large the incisions shown in Fig. 51 may be made. By dissecting up the flaps E A B F and G C D H they may be slid towards each other, the resulting scar taking the form of an H. In excising nævi

NAILS, DISEASES OF

air to cutaneous nævi is simple. No anæsthetic is necessary. A small piece of wool held in forceps is dipped in the liquid air and applied to the surface of the nævus for a few seconds.

Carbon dioxide snow.—For freezing a nævus no anæsthetic is necessary. The stick of solid carbon dioxide, held in a piece of cotton-wool, is applied to the tumour and kept in contact for about 40 seconds—the time of application, however, depending upon the amount of pressure. A blister appears subsequently, but very little scarring results if the application is not too prolonged.

LYMPHATIC NÆVUS

Lymphatic nævi are localized collections of dilated lymphatic vessels (lymphangioma). Sometimes much dilatation of the spaces takes place (cavernous lymphangioma, cystic hygroma). These tumours are usually circumscribed, and when subcutaneous present features similar to those of hæmangiomata, except that the skin is pale over them and there is no bluish hue showing through. Lymphatic nævi occur anywhere on the surface of the body, but especially on the forehead, tongue, and inner surfaces of the lips or cheeks. A large diffuse growth may cause much increase in size of the tongue (macroglossia) or of the lip (macrocheilia). The cavernous form occurs especially in the neck, axilla, and groin.

The **treatment** of lymphangiomata of the subcutaneous tissue and of the mucous membrane of the cheeks or lips is carried out as described above. That of cavernous lymphangiomata of the neck may prove to be a very serious undertaking. They are liable to be diffuse, and at operation the dilated spaces are found deep in the posterior triangle, surrounding the large vessels of the neck. Indeed, sometimes their removal is quite impossible. If imperfectly removed, uncontrollable lymphorrhagia may result, from which the patient dies. Fortunately, lymphangiomata of the neck and axilla tend to disappear spontaneously, or to shrink after the attacks of inflammation to which they are liable.

C. A. PANNETT.

NAILS, DISEASES OF.—Diseases of the nails may be due to (1) congenital defect, (2) local disturbances of nutrition of unknown cause, (3) local infection or traumatism, (4) various skin diseases of which the nail changes form a part, (5) general systemic and nervous dis-

eases in which affections of the nails result from malnutrition or toxæmia, or from neurotrophic and circulatory disturbances. Owing to the fact that the same changes in the nails are common to many different diseases, their classification and diagnosis generally depend upon the presence of concurrent symptoms elsewhere, e.g. in the cutaneous or the nervous system.

1. **Congenital affections.**—One or more nails may be missing, or there may be supernumerary nails.

2. **Affections due to local disturbances of nutrition of unknown cause.**—Several of these may be congenital, and a few hereditary and familial. *White spots (leuconychia)* are said to be caused by slight injuries and the inclusion of air in the nail substance.

Curved nails (onychogryphosis).—The great toes are principally affected. The nail is enormously hypertrophied and becomes curved upon itself as it increases in length, so as to resemble a ram's horn. The condition has been attributed to the wearing of tight boots, but often occurs without any apparent cause. Treatment consists in paring away or removing the affected nails.

Spoon-shaped nails (koilonychia).—The sides and free edges of the nail are raised from the nail-bed so that the nail becomes concave. This condition may be due to eczema or psoriasis, or may occur as an independent condition.

Splitting of the nails (onychorrhexis).—The nails are brittle and become reeded or fluted from longitudinal striation and splitting. Such nails may occur in psoriasis, eczema, lichen planus, or gout, or without any apparent cause. Treatment, if required, is by painting with collodion.

Separation of the nail (onychoschizia).—Partial or complete separation of the nail from its bed without any underlying keratosis or accumulation of scales is a rare condition. The cause is unknown, but it is said to occur in neurotic subjects. A similar affection has been described as the "egg-shell" nail.

Tylosis is a rare form of hypertrophy in which the thickened nail is raised from its bed by subungual hyperkeratosis. It may affect many members of a family.

3. **Local infection and traumatism.** *Septic onychia.*—Small abscesses occur at the edges of the nails as the result of staphylococcal infection, e.g. from nail-biting, or of streptococcal infection occurring in association with

NAILS, DISEASES OF

NASAL POLYPUS

impetigo elsewhere, or independently, and giving rise to perionychia and whitlow.

Parasitic or hyphomycetic onychia.—The onychomycoses consist of ringworm and favus due to the *Trichophyton endothrix* or *endoctothrix* and *Achorion schönleinii* respectively, and are described in the articles devoted to these subjects.

Syphilitic onychia.—This includes primary chancre of the nail and the onychia and perionychia of both congenital and acquired syphilis.

Traumatic onychia.—The nails may be injured as the result of accidental violence or by constant exposure to radium or X-rays; they may be worn away by constant scratching in pruritic skin affections, or be injured by chemical substances employed in certain trades, e.g. the bichromates used by french-polishers, aniline dyes, etc.

4. Various skin diseases of which the nail changes form a part. *Ecze-ma.*—There are no changes in the nails peculiar to eczema, but the nails may be pitted, ridged, or grooved in a transverse or longitudinal direction, opaque and darkened in colour, and in rare cases may be shed.

Psoriasis.—The nails are more commonly affected in this disease than in eczema. They show minute punctate pitting as in that disease. In other cases a patch of psoriasis is formed beneath the nail, generally at the free end, but occasionally at the lateral edges or at its root, and the nail plate is then opaque and is lifted up and distorted by the subjacent scales. (See also PSORIASIS.)

Other skin diseases.—In pityriasis rubra the nails are often completely shed; in pityriasis rubra pilaris they are sometimes thickened and striated; in epidermolysis bullosa, pemphigus, Darier's disease, acanthosis nigricans, alopecia areata, and lichen planus they may undergo atrophic changes similar to those already described.

5. General systemic and nervous diseases.—Acute pyrexial and debilitating diseases sometimes give rise to the formation of transverse furrows, thus marking the date of the illness. Longitudinal striation, opacity, and brittleness result from gout, and likewise occur as senile changes. Enlargement of the nails with clubbed fingers is common in thoracic disease. A few cases of hæmorrhage into the roots of all the finger-nails, of unknown origin, have been recorded. Neuritis, anæsthetic leprosy, syringomyelia, hemiplegia, tabes dorsalis, Raynaud's

disease, scleroderma, chronic tuberculosis, and other nervous and circulatory disturbances may be accompanied by dystrophy of the nails or render them liable to septic infection as the result of malnutrition and trophic changes.

S. E. DORE.

NARCOLEPSY (see SLEEP, DISTURBANCES OF).

NASAL CATARRH (see CORYZA).

NASAL POLYPUS.—Polypi occur either singly or in large numbers, and may be either unilateral or bilateral. Disease of the ethmoidal cells, of the middle turbinal bones, or of the maxillary antra is the usual cause.

Symptomatology.—There is always some nasal obstruction, which may vary from only a very slight difficulty in breathing through the affected nostril to an obstruction which is complete. Loss of smell is also complained of; it may be partial or absolute, according to the amount of polypoid tissue present. Nasal and postnasal discharge occurs in all cases, and is usually muco-purulent; it often collects in the postnasal space and forms crusts which cause a great deal of discomfort and are very difficult to dislodge. Occipital or frontal headache is usually experienced; it may be slight or very severe. In cases of long standing, broadening of the nose may result. Pain, tenderness, or actual swelling may appear over the frontal sinuses or the antra, due to the retention of the discharge in these sinuses by blocking of their outlets. Acute symptoms due to the retention of discharge in the ethmoidal cells may also occur.

Aural, pharyngeal, and laryngeal **complications** supervene when the disease has been present for a considerable time, the commonest being middle-ear suppuration, deafness, tinnitus, chronic pharyngitis, laryngitis, and laryngitis and pharyngitis sicca.

Diagnosis.—On examination by anterior rhinoscopy, polypi appear as grape-like masses. When only a few small growths are present, they can easily be recognized in the vicinity of the middle turbinal bones. When, however, the nostril is completely blocked, the polypi are seen lying between the septum and outer wall of the nose as a pale fleshy mass. They are distinguished from an enlarged anterior end of the middle or inferior turbinal bone by their softness and by the ease with which they can be moved about with a probe—an enlargement of an anterior end of a turbinal bone

NASO-PHARYNGEAL OBSTRUCTION

being immobile and firm on pressure with the probe, and pink in colour.

It should be remembered that occasionally there are found in the nostril polypi whose seat of origin is in the maxillary antrum; their pedicles will be found to reach the antrum by passing up under the anterior end of the middle turbinal bone. In a certain number of cases they are seen in the postnasal space by posterior rhinoscopy.

Treatment.—When a single polypus is present it can be removed with ease by a snare after the nostril has been packed with a 10-per-cent. solution of cocaine. The base of attachment should not be allowed to remain, but should be punched away, otherwise recurrence will take place in quite a short time. When the polypi are multiple it is advisable to give a general anæsthetic and remove the masses with a pair of Luc's forceps. At the same operation the anterior end of the middle turbinal bone should always be taken away, and any diseased ethmoidal cells opened up.

The importance of reporting for examination at regular intervals must be impressed upon the patient, as recurrences are very likely to take place. It is well to advise him to report every seven days for the first month, after which the interval can gradually be increased.

Hæmorrhage serious enough to call for active treatment is rare; plugging of the nostril with ribbon gauze soaked in hydrogen peroxide usually suffices to stop it, but should it be exceptionally severe the postnasal space may have to be plugged as well.

An alkaline douche, morning and evening, should be prescribed as a routine treatment. The douching should be started about four days after the operation.

When crusts form, a spray of 5-per-cent. menthol in paroline should be used after the nose has been douched.

G. N. BIGGS.

NASAL SEPTUM, DEFLECTIONS OF
(see NASO-PHARYNGEAL OBSTRUCTION).

NASAL SINUSES, DISEASES OF (see
SINUSES, ACCESSORY AIR, DISEASES OF).

NASAL SPUR (see NASO-PHARYNGEAL OBSTRUCTION).

NASO-PHARYNGEAL OBSTRUCTION.

—The causes of naso-pharyngeal obstruction are many, and include adenoids, rhinitis in its various forms, syphilitic, leprosy, and tuberculous disease of the nose, foreign bodies, nasal

polypi, spurs and deflections of the septum, new growths, hæmatomata, rhinoliths, rhinoscleroma, and malformations. For the most part these are described under their several heads. Hypertrophic conditions of the turbinal bones, deflections of the septum, and nasal spurs are considered here.

General effects of naso-pharyngeal obstruction.—These are so well known as to need only a brief reference. Among the commonest are loss of memory and digestive troubles, the latter being especially met with in cases of sinus suppuration in which the patient is constantly swallowing mucus.

In some cases night-sweats occur, and a general feeling of weariness on waking in the morning. Deformities of the chest and underdevelopment both general and mental (aprosia) are frequent results.

Secondary disorders of the ear and the respiratory tract are almost always present in protracted naso-pharyngeal obstruction, the commonest being deafness, middle-ear suppuration, Eustachian obstruction, acute and chronic laryngitis, tracheitis, and bronchitis. Dental caries (q.v.) is found nearly always, and is probably the result of the chronic mouth-breathing. Reflex symptoms such as incontinence of urine, asthma, neuralgia, and stammering are met with in a certain proportion of cases. When suppuration is present in the nose, general symptoms such as pallor and loss of weight always occur sooner or later from the constant absorption of toxins. None of these secondary disorders can be dealt with satisfactorily until the nasal obstruction, its cause, has been correctly treated.

HYPERTROPHIC CONDITIONS OF THE MIDDLE AND INFERIOR TURBINAL BONES.—The cause in almost all cases is either repeated attacks of acute rhinitis, or chronic rhinitis. Sometimes hypertrophy occurs in the early stages of suppuration in the nasal accessory sinuses, when it is due to constant irritation of the mucous membrane by the passage of pus over its surface. This variety is usually unilateral, unless the patient has bilateral sinus suppuration. Excessive smoking is also a probable cause, and the same may be said of alcoholic excess.

ENLARGEMENT OF THE ANTERIOR END OF THE MIDDLE TURBINAL BONE.—The commonest symptom is a constant or intermittent headache, sometimes very severe; it is usually situated in the frontal region, but may be occipital only. Attacks of acute rhinitis are

NASO-PHARYNGEAL OBSTRUCTION

usual, and the consequent swelling of mucous membrane renders the headaches more severe and persistent. There is often loss of smell.

The nasal obstruction is usually slight, unless the inferior turbinal bone is also hypertrophied.

Diagnosis.—The enlarged anterior ends of the middle turbinal bones are easily detected by anterior rhinoscopy. The posterior ends of this bone are very rarely affected. When the inferior turbinal bones are much enlarged it may be necessary to apply cocaine to them before the middle turbinal bones can be brought into view.

ENLARGEMENT OF THE ANTERIOR END AND BODY OF THE INFERIOR TURBINAL BONE.—Obstruction in varying degrees that depend upon the amount of enlargement is the symptom which causes the patient most discomfort. A mucous or muco-purulent discharge is always present, and may either emerge from the anterior nares or the postnasal space. Headache occurs in some cases. At night the obstruction becomes more severe and the patient is compelled to resort to mouth-breathing, with the result that the mouth is always dry and unpleasant on waking in the morning. During attacks of acute rhinitis, which are common and recur repeatedly, the symptoms are always much increased in severity.

If the enlargement has been present for a considerable time, secondary symptoms such as deafness, tinnitus, and attacks of hoarseness appear and add greatly to the patient's discomfort.

No difficulty is experienced in making a diagnosis, as the enlargement is easily detected by anterior rhinoscopy.

ENLARGEMENT OF THE POSTERIOR END OF THE INFERIOR TURBINAL BONE.—The most characteristic symptom is the advent of nasal obstruction whenever the recumbent position is assumed for any length of time; it is therefore most troublesome and persistent at night and passes off during the day. The mouth is always dry in the morning, and there is usually a chronic postnasal discharge which is sometimes very difficult to dislodge on waking. Crust-formation in the postnasal space occurs in some cases.

Such secondary symptoms as temporary attacks of deafness and tinnitus and laryngitis usually occur sooner or later.

The enlarged posterior ends can be seen by posterior rhinoscopy, and also by anterior

rhinoscopy when there is no enlargement of the anterior end of the inferior turbinal.

Treatment of obstruction due to enlargement of the turbinal bones.—This consists in all cases in removal of the affected parts. Care must be taken to remove only those parts which are actually enlarged. In no circumstances is it justifiable to remove the whole bone, for atrophic rhinitis is apt to result, and the patient's condition to be much worse than before the operation, owing to the crust-formation and to the dry condition of the pharynx and larynx which inevitably follows.

For three or four days a preliminary nasal douche of normal saline, morning and evening, is advisable. The operation may be carried out under local or general anaesthesia, but if the practitioner is not expert at intranasal surgery a general anaesthetic is for obvious reasons preferable. When a local anaesthetic is employed the patient should be sitting up. Ten-per-cent. cocaine is the best anaesthetic; it should be applied by packing the nostrils with gauze soaked in the solution. A word of warning is necessary when cocaine is employed either alone or in conjunction with a general anaesthetic. It causes shrinking of the areas to which it is applied, and the practitioner should therefore always remove a little more of the turbinal bone than appears necessary at the time, lest when the effects of the cocaine pass off it be found that the obstructing parts have not been completely removed and that a second operation is called for.

Anterior end of the middle turbinal bone.—A nasal speculum having been inserted, the light from the mirror or head light should be focused upon the anterior end of the middle turbinal bone, and as much as is necessary of the bone should be removed by morcellment with a pair of Luc's ethmoidal forceps.

Great care and gentleness must be exercised during operative procedures in this region, otherwise serious injury to the orbit or even damage to the cribriform plate may result.

The nostril should be packed gently with a strip of gauze soaked in sterile paroline to control the hæmorrhage; the paroline will prevent the gauze from becoming adherent to the wound surface as it dries. The plug should be removed in twenty-four hours, and re-packing is practically never necessary.

Anterior end and body of the inferior turbinal bone.—The light should be focused on to the affected parts, and as much as is necessary of

NASO-PHARYNGEAL OBSTRUCTION

the anterior end and body of the inferior turbinal bone should be removed piece by piece. Never should the whole of the anterior end of the inferior turbinal bone be removed, but only enough of the surface adjacent to the septum and of the lower border to establish an airway of about $\frac{1}{8}$ in. between the turbinal bone and the septum and between the turbinal bone and the floor of the nose. A plug of flat rubber sheeting should then be inserted into the affected nostril; this will effectually prevent any adhesions forming between the turbinal bone and the septum. The plug should be removed in four or five days.

Posterior ends of the inferior turbinal bone.

—A general anæsthetic should always be administered in these cases, as cocaine causes the enlargement, which is always one of mucous membrane and not of bone, to shrink so that its removal is almost impossible. A Lake's turbinotome (Fig. 52) should be used. It is

hydrogen peroxide (10 vols.) usually arrests it quickly.

If rubber plugs have been inserted the anterior nares can be cleansed by the insertion of a few drops of peroxide of hydrogen, followed by gentle irrigation with normal saline solution, morning and evening, or more often if necessary.

Seven days after the operation the patient may use an alkaline nasal douche morning and evening, followed, if there is any crusting, by a spray of 5-per-cent. methyl salicylate in paroline. This treatment should be carried out for about three weeks. Aspirin in 10-gr. doses will usually relieve any neuralgia.

DEFLECTIONS OF THE SEPTUM.—Deflections may result from injury or be congenital in origin, and may involve the cartilaginous or bony septum, or both. The septum may bulge in either direction or may be S-shaped and

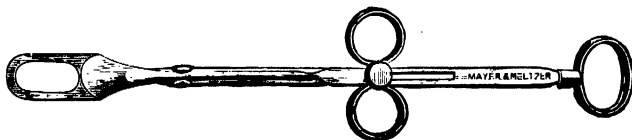


Fig. 52.—Lake's turbinotome.

passed along the floor of the nose until it is felt to impinge upon the posterior wall of the naso-pharynx; the blade is opened and the instrument drawn forwards against the outer wall of the nose, until the posterior end of the inferior turbinal bone engages the ring; the blade is then pushed home and the instrument withdrawn. If any difficulty is experienced, the procedure may be facilitated by inserting a finger into the patient's naso-pharynx and guiding the ring over the posterior end by touch.

When a snare is used the loop can be guided over the posterior end of the turbinal bone in the same manner.

Brisk hæmorrhage always occurs for a few minutes, but rarely calls for any local measures such as plugging.

After-treatment.—In all cases a hypodermic injection of morphia should be given, as it helps to control the hæmorrhage, and a piece of lint, which should be kept constantly moistened with ice-cold water, can be applied to the bridge of the nose.

If there is much oozing, the insertion into the nostrils of a piece of wool soaked with

obstruct both nostrils. Children are quite commonly affected.

Nasal obstruction, which is the main symptom, may be slight and limited to one nostril, or both nostrils may be obstructed partially or completely. Epistaxis occurs in some cases, and loss of smell and headache are usually present if the deflection presses on the middle turbinal bone. The patient usually suffers frequently from colds, during which all the symptoms become greatly exaggerated.

Later, hypertrophy of the middle and inferior turbinal bones usually occurs, and is most pronounced in the nostril on the concave side of the deflection.

Secondary aural, laryngeal, and general symptoms such as deafness, laryngitis, loss of weight, and a general feeling of malaise follow sooner or later in most cases.

On examination, the deflection will be seen bulging into one nostril, whilst in the other nostril a corresponding depression will be detected.

Treatment.—Submucous resection is the only treatment that provides complete relief from the symptoms. It is an operation which,

NEEDLE-FRAGMENTS IN THE HAND, EXTRACTION OF

if carried out properly, gives excellent results, but, if the septum is badly perforated, may make the patient very much worse. A practitioner who has not had ample experience with this operation is strongly advised not to attempt it.

SPURS AND CRESTS OF THE SEPTUM.—These may occur in any position, but most frequently they run upwards and backwards, and involve both the cartilaginous and bony part of the septum. The symptoms are identical with those produced by deflections. Those which are local are usually limited to one nostril, unless hypertrophy of the inferior or middle turbinal bones is present. On examination, the crest or spur can readily be identified as a local projection from the septum. There is no corresponding concavity in the other nostril, as in the case of a deflection of the septum.

Treatment.—Operation is the only satisfactory treatment, and the same remarks apply as in deflections of the septum.

G. N. BIGGS.

NASO-PHARYNGITIS (see CORYZA).

NAUSEA (see STOMACH, FUNCTIONAL DISORDERS OF).

NECROBIOSIS (see UTERUS, NEW GROWTHS OF).

NECROPSY (see POST-MORTEM EXAMINATION IN MEDICO-LEGAL CASES).

NECROSIS OF BONE, ACUTE (see OSTEO-MYELITIS).

NECROSIS OF JAW (see ORAL SEPSIS).

NEEDLE-FRAGMENTS IN THE HAND, EXTRACTION OF.—The removal of fragments of needles which have penetrated into the hand or finger is sometimes a very tedious procedure, but it is one that may be faced with confidence if undertaken methodically. Never should the attempt be made without previous skiagraphy, unless the needle can be seen or felt indubitably by the palpating finger. There is no necessity for a complicated localizing technique, nor for the taking of X-ray plates. A far more useful impression of the exact situation of the needle will be obtained by a screening performed by the surgeon himself. He should observe the position of the needle in relation to bony landmarks which he can recognize at operation, and note the change of position when the hand is turned over from

the flat to the edge-on position. He will thus be able to decide whether to operate from the palmar or the dorsal surface. He will also be able to determine whether the needle changes its position with flexion and extension of the fingers or wrist; such a movement indicates that the needle is fixed in a tendon, and will exactly localize it. Finally, he should mark with ink the position of the carefully centred needle, on the surface from which he will operate, with the hand absolutely flat and parallel with the screen. The screen examination should take place at a time as close to the operation as possible, for sometimes a needle will move its position with the movements of the fingers; this is especially true if it should be loose in the anterior carpal sheath. If the surgeon cannot carry away a vivid memory-picture of what he has seen, negatives must be made, but they should not be a substitute for his personal screen examination.

It is not wise to attempt these operations under local anaesthesia. General narcosis is more satisfactory both to patient and surgeon. A tourniquet should be applied to the upper arm and the dissection made deliberately. The most difficult situations in which to find a needle are the palm and the front of the wrist. When the needle is in the central portion of the palm the incision should be made directly over the skin mark, access to the needle being gained by careful dissection between the various structures of the palm. When the needle is in the thenar eminence, make the incision on the radial side of the metacarpal bone of the thumb. Cut through the deep fascia and find the radial border of the abductor pollicis. Then insinuate the index finger between this muscle and the opponens pollicis. With the thumb on the skin externally it will be possible to palpate the abductor and flexor brevis pollicis muscles, and a needle in them will be detected with certainty. If it is not found here the pulp of the finger is turned backwards to feel the opponens pollicis. It may be necessary to detach the opponens from the metacarpal shaft and enter the interval between this muscle and the first dorsal interosseous muscle. By these manœuvres any needle in the thenar eminence will be found without difficulty. When the needle is in the hypothenar eminence the incision should be made on the ulnar side of the fifth metacarpal bone and a corresponding intermuscular dissection made. The tissues in the neighbourhood of

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a needle are stained black. This fact is of help in the search. An incision should not be made on the palmar surface of the pulp of the finger. A needle in this situation may be reached by a lateral cut. When the needle is in front of the wrist the incision is made directly over the skin mark. The annular ligament may require division in order to reach it. When a needle is searched for deliberately in the exsanguinated limb after a careful screen examination it is very seldom that one fails to find it. But in the rare event of the attempt proving unsuccessful the wound should be sutured with the eye half of a needle sewn in the bottom of it. A subsequent screen examination will show the relation of this foreign body of known position to the one sought for, and will reveal the true situation of the latter; or the second search may be made on the X-ray couch with the aid of the fluorescent screen—a procedure, however, which does not allow of such careful operating or aseptic technique.

C. A. PANNETT.

NEPHRITIS.—This term, used without qualification, denotes a bilateral non-suppurative inflammation of the kidneys, of acute, subacute, or chronic course, involving the different tissue elements, parenchyma, connective tissue, and blood-vessels, in varying degree. Associated with these changes is an impairment more or less severe of the renal function, with a train of symptoms mainly dependent thereupon.

The current classification of the various forms of nephritis, based on morbid anatomy, is admittedly unsatisfactory, but in the present state of our knowledge a re-grouping more in accordance with clinical findings and the results of function testing is hardly possible. It is customary to speak of nephritis in terms of the tissue principally involved. Thus we have parenchymatous nephritis, interstitial nephritis, and the arterio-sclerotic kidney; but between the large white kidney with predominant parenchymatous change and the small contracted forms of interstitial nephritis every variation is met with. Glomerular changes necessarily affect the tubules, interstitial overgrowth produces its results on the parenchyma, and vascular degeneration influences all, with the result that cases of nephritis not infrequently fail to conform to type. Further, an attack of acute inflammation may supervene on the chronic form, and the picture becomes still more complex. The

chief forms of nephritis may be set down thus:—

1. ACUTE DIFFUSE NEPHRITIS.
2. CHRONIC PARENCHYMATOUS NEPHRITIS.
3. CHRONIC INTERSTITIAL NEPHRITIS:
 - (1) Primary Granular Kidney.
 - (2) Contracted White Kidney.

The descriptions of these forms of nephritis are prefaced by a brief discussion of modern methods of investigating the renal function, and are followed by sections on—

4. WAR NEPHRITIS.
5. RENAL SYPHILIS.
6. UREMIA.

INVESTIGATION OF THE RENAL FUNCTION

Complete examination of a case of nephritis includes an investigation of the functional activities of the kidneys. Medical literature of recent years has been especially rich in the methods and results of such investigation, and it is to be regretted that much of this work is too complex for general clinical application. Hugh MacLean has made valuable researches on war nephritis and, later, on the nephritis of civilian practice, and I gladly acknowledge my indebtedness to his works. He has recommended three tests as of proved value in the determination of kidney function, as follows:—

i. **The blood-urea.**—The healthy individual has 15–40 mg. of urea in 100 c.c. of blood. With this concentration he is able to excrete 25–30 grm. of urea daily in the urine. In some cases of acute nephritis, but typically in chronic interstitial nephritis, a higher blood concentration is necessary to bring about the same result, and nitrogen retention occurs. Blood-urea concentrations of more than 40 mg. per 100 c.c. of blood are abnormal, but in severe cases there may be 200–300 mg. In acute nephritis persistent high readings indicate a progressive lesion, and in chronic interstitial nephritis they affect the prognosis adversely in proportion to the amount of retention. Such cases belong to the azotæmic group. In chronic parenchymatous or hydræmic forms, nitrogen retention is not present and blood-urea figures are within normal limits. The technique employed consists in the conversion of the blood urea into ammonium carbonate by means of a ferment, urease. In the presence of an alkali, ammonia is liberated and is passed through a standard acid by means of a current of air.

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The acid is subsequently titrated with $n/100$ caustic soda. The method is at present restricted to hospital practice, and works on chemical physiology and pathology should be consulted for a detailed description.

ii. **MacLean's urea concentration test.**—The estimation of the urinary urea in ordinary circumstances is of little value, since it has been shown that wide variations occur in health; thus, in hospital patients in bed an average percentage is 1.6. Attempts have been made

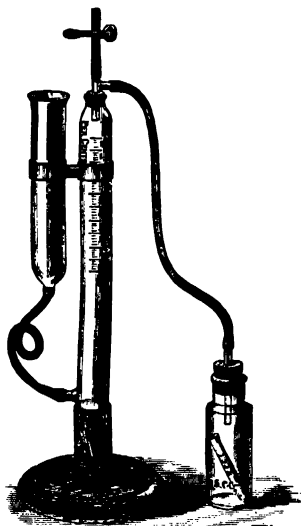


Fig. 53.—Gerrard's ureometer.

to estimate the urinary urea on a diet of known nitrogen content, a renal test meal. The procedure is laborious and the results have not shown uniformity. To overcome these difficulties MacLean has devised a test for estimating the power of the kidney to concentrate urea. In his hands it has proved the most reliable of all tests for renal function. The technique, which is simple, is as follows:—

(a) The patient empties his bladder and immediately afterwards takes 15 gm. of urea dissolved in 100 c.c. of water.

(b) One hour later he passes water. The sample is kept and measured.

(c) Two hours after taking urea he again passes water, the sample being measured and retained for urea estimation.

The second specimen is used in preference

to the first, because the urea may give rise to a certain degree of diuresis, especially in patients who have been imbibing large quantities of fluid before the test. As a rule, the diuresis has spent itself in the first specimen, but occasionally it continues into the second hour, and, if this be so, the specimen at the end of the third hour should be estimated for urea. In a case in which there are no other signs of renal disease, the passage of large quantities of urine of low urea concentration depends on diuresis and does not indicate renal disease. The quantity of urine passed per hour should not average more than 150 c.c. When quantities of 350–600 c.c. are passed, any tendency to low urea concentration should be attributed to the diuresis. The urea is estimated by means of Gerrard's ureometer (Fig. 53), or some modification of it.

The results show that healthy individuals respond to the dose of urea by concentrating 2–4 per cent. Figures below 2 per cent. indicate renal impairment; moderately severe cases can only concentrate 1.4–1.5 per cent.

iii. **The diastase reaction.**—Normal urine contains a certain definite amount of a diastatic ferment obtained from the pancreas through the circulating blood. Its measurement is made use of as a test for renal function. The test consists in estimating the amount of starch, which is converted into dextrin by a given volume of the urine, iodine being used as an indicator. A specimen of the twenty-four hours' excretion is necessary. The following solutions are required:—

1. A solution of 0.1 per cent. soluble starch in a small amount of boiling water, cooled and made up to 100 c.c. volume.

2. A 0.9-per-cent. solution of sodium chloride.

3. A 10-per-cent. solution of iodine.

Five test-tubes are taken and numbered 1 to 5; the following amounts are added to each of the tubes by means of a 1-c.c. pipette graduated in hundredths:—

Tube No.	Urine c.c.m.	Normal saline c.c.m.
1	1	0
2	0.6	0.4
3	0.3	0.7
4	0.2	0.8
5	0.1	0.9

To each tube 2 c.c.m. of starch solution are added. The tubes are rapidly shaken and placed in an incubator or water-bath at 37° C. for exactly thirty minutes. They are then removed, and each is filled to within an inch of the top with cold water in order to stop the ferment action. One drop of

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the iodine solution is now added, beginning at tube No. 5. On shaking, it may be found that tubes No. 5 and 4 remain blue, while No. 3 gives a faintly pink tinge, indicating that this tube contained just sufficient urine to convert 2 c.c. of soluble starch into dextrin in half an hour. The unit is obtained by dividing the amount of starch by the quantity of urine needed for the conversion ; thus, $\frac{2}{0.3} = 6.6$ units.

If on addition of the iodine no colour develops owing to the large amount of urine present, an extra drop or so will cause the colour to appear.

Healthy individuals give diastase readings varying between 6 and 30 units. The test should be performed in conjunction with the urea concentration test, when, generally speaking, a low diastase reading is associated with a failure to concentrate urea.

Other tests.—The estimation of the urinary chlorides will give information as to salt retention. Here again very wide variations occur in health, so that it is necessary to put the patient on a diet of known salt content for some days before giving a definite dose of salt with subsequent estimation of the urinary chlorides. The method is elaborate, and the results so far do not justify its inclusion here. Further, any material degree of salt retention is rendered obvious by the resulting oedema.

The excretion of various dyestuffs has also been made use of, the time between ingestion or subcutaneous injection of the dye and its subsequent appearance in the urine being noted. The phenolsulphonaphthalein test of Rowntree and Geraghty is the best of these, but the results have proved of less value than those of the tests described above. Its sphere of usefulness is probably greater in surgical affections of the kidneys.

Conclusions.—The three tests recommended by MacLean give valuable information as regards the diagnosis and prognosis in nephritis. The results can be tabulated thus :—

	Acute nephritis	Chronic parenchy- matous nephritis	Chronic interstitial nephritis
BLOOD-UREA	High	Normal or low	High
UREA CON- CENTRA- TION	Low	Normal	Low
DIASTASE CON- TENT	Variable	Usually normal	Low

The results should invariably be appraised in the light of clinical examination, to which they are complementary. The complete investigation of a case of nephritis takes cognizance of the following points :—

General condition of patient.

Presence or absence of albuminuria.

Presence or absence of casts.

Presence or absence of oedema

Blood-pressure.

Position of the heart's apex beat.

Condition of vessels.

Results of urea concentration test.

Urinary diastase.

1. ACUTE DIFFUSE NEPHRITIS (ACUTE BRIGHT'S DISEASE)

An acute non-suppurative inflammation of the kidneys due to the action of a chemical or bacterial poison.

Under this heading will be included those forms of acute nephritis variously known as acute glomerular, acute tubular, and acute interstitial nephritis, the clinical manifestations being essentially the same.

Etiology.—The causes which may give rise to this condition are—

(1) **Acute febrile disorders.**—In this connexion the specific fevers are the most important, and among them scarlet fever stands pre-eminent. It is noteworthy that the incidence of nephritis bears no relationship to the severity of the disease. Next in importance comes diphtheria, whilst less common causes are measles, rubella, smallpox, chickenpox, cerebro-spinal meningitis, malaria, cholera, yellow fever, typhus fever, the enteric-group disease, and dysentery. Mention should also be made of the toxic albuminuria of febrile disorders, the transitory nephritis of French writers. In these cases the damage to the renal parenchyma is sufficient to allow the escape of protein, but insufficient to give rise to symptoms characteristic of nephritis. The great majority recover completely, but in a few the lesion progresses to a true nephritis. This is seen occasionally in typhoid fever, diphtheria, and pneumonia ; more frequently in influenza and scarlet fever. Of other febrile states which may give rise to acute nephritis, tonsillitis is the most important.

(2) **Chemical poisons.**—Examples of these are turpentine, cantharides, potassium chlorate, carbolic acid, corrosive sublimate, and alcohol. Alcohol is probably a predisposing rather than a direct cause of acute nephritis.

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(3) **Exposure to damp and cold.**—The exact part played by these agencies is obscure. Acute nephritis undoubtedly occurs after such exposure, and especially among those whose daily occupations subject them to extremes of temperature. Probably in all cases some antecedent infection was present, the exposure being merely a determining factor.

(4) **Certain lesions of the skin.**—Extensive superficial burns and some chronic skin diseases are occasional antecedents.

(5) **Pregnancy.**—During gestation a previously latent nephritis may be brought to light, or the nephritis may originate *de novo* as a result of the toxæmia of pregnancy.

(6) **Syphilis and tuberculosis.**—Syphilis in its secondary stage and, very rarely, acute tuberculosis will produce nephritis. Syphilitic nephritis receives notice later (p. 353).

(7) **Conditions of active service** have proved to be a potent cause of acute nephritis, the main features of which are dealt with at p. 352.

Predisposing and contributory causes are—

(a) **Age.**—Acute nephritis occurs at any age, but is commonest in the young owing to their greater liability to the specific fevers.

(b) **Sex.**—Males are affected somewhat more frequently than females, possibly owing to greater exposure and severer forms of physical work. The incidence is especially high among those whose work exposes them to extremes of temperature.

Morbid anatomy. Macroscopic appearances.—The kidneys may be normal in size and show nothing pathological to the naked eye; commonly, however, they are somewhat enlarged and tense. The colour varies with the amount of blood in the organ and with the presence of hemorrhages and fatty changes. The latter indicate a late form of the disease and a transition process to the subacute or chronic type. The cut section bulges and shows a swollen cortex, whose pale colour is in strong contrast with the deep red of the congested pyramids. The glomeruli may appear as red dots. Occasionally the changes seen are mainly those of chronic nephritis with a terminal acute exacerbation supervening.

Microscopic appearances.—In the majority of cases, and particularly in the scarlatinal form, the glomeruli are chiefly affected. The tufts are swollen and come to fill the capsule more completely. Increased cellularity is present owing to the appearance of wandering cells and endothelial proliferation. Hyalin

thrombi may be seen in the vessels. At a late stage adhesions are formed between the tuft and the capsular wall; these may become organized. A variable amount of blood with leucocytes and fibrin fills up the lumen of the capsule. The tubules almost invariably show some change, owing partly to interference with the blood supply through the glomerulus, and partly to direct toxic action on the cells. Various stages of degeneration and desquamation of the lining membrane are present. Blood and leucocytes are also seen in the tubules. Associated with these epithelial changes, inflammatory reaction occurs in the stroma and is revealed by the presence of wandering cells and by oedema causing a wider separation of the glomeruli and tubules.

The brunt of the damage sometimes falls upon the connective tissue. Councilman has described such cases from histological investigation among children in fever hospitals. The condition is one of acute interstitial nephritis and may occur in the absence of any symptoms referable to the kidneys.

Changes in the vessels become apparent after about six weeks' illness and indicate a transition to subacute and chronic types.

Symptomatology.—Two distinct types of onset can be recognized. One, the acute type, is ushered in by symptoms such as chills, rigors, convulsions in children, headache, pain in the loins, vomiting, constipation, and oedema. The other form appears insidiously and is manifest by physical signs rather than by symptoms, puffiness round the eyes and the passage of blood in the urine attracting attention. The latter mode of onset is seen typically in association with the specific fevers.

The urine.—The quantity is usually reduced to a few ounces in the twenty-four hours, or there may be complete suppression. The specific gravity is raised and varies between 1025 and 1035; the reaction is strongly acid; the odour is characteristic and resembles that of beef tea. Albumin is present in considerable quantity. The quantity of blood varies from the trace required to produce a smoky tinge to large amounts giving a deep red coloration. The deposit is heavy and muddy-looking from admixture with altered blood. Microscopically, cells from the urinary passages and various forms of casts, blood, hyalin and epithelial, are present. The daily excretion of the solid constituents is diminished, though the percentage of urea in any given specimen may be high. The passage of the urine is often

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painful, and there may be a frequent desire to micturate.

Œdema.—Most patients develop œdema of greater or less amount, but this bears no relationship to the severity of the nephritis; indeed it may be entirely absent with very advanced renal destruction. The situation of the œdema is important and characteristic, the loose tissues round the orbit and the ankles being the parts first affected. The scrotum and penis are often involved, and finally general anasarca may develop. Ascites and hydrothorax are seen occasionally. The greatest amount of dropsy is seen in cases where the glomeruli are principally affected, as in the scarlatinal type. Œdema of the larynx, lungs, brain, and spinal cord may occur.

Circulatory system.—A moderate rise of blood-pressure—20 mm. Hg or more—may be observed after a few days, and associated with it there may be headache of a severe and throbbing character. Persistent high blood-pressure with cardiac hypertrophy occurs after about six weeks' illness, and invariably indicates the presence of a chronic lesion. Heart failure may result from this. Pericarditis may occur as a terminal event.

Respiratory system.—Bronchitis may supervene; it was a prominent feature of War nephritis. Lobar and broncho-pneumonia are rare, as also is acute pleurisy. They are apt to end fatally. Acute pulmonary œdema forms at times part of the general dropsy, but may occur in its absence. Apart from gross intrathoracic disease, dyspnoea may be present as a manifestation of uræmia.

Alimentary system.—Nausea and vomiting may appear as initial symptoms. In the later stages vomiting should suggest uræmia. The tongue is furred and the breath somewhat fœtid. Constipation is almost invariable, though diarrhœa is occasionally seen in children. General peritonitis is a very rare and fatal event.

Fever.—The temperature is often raised to 101° or 102° F. for the first few days, but it is not a constant feature and the disease may run an afebrile course.

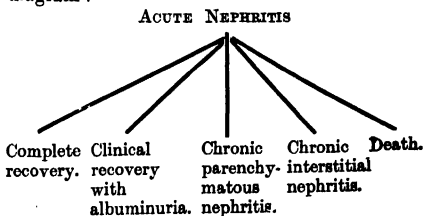
The blood.—The changes characteristic of secondary anæmia are usually present. Epistaxis and hæmorrhages from other mucous membranes may appear in association with the raised blood-pressure. Chemically there is usually an increase in the blood urea, which may amount in very severe cases to 200–300 mg. per 100 c.c. of blood, instead of the 15–40 mg. present in health. Subconjunctival hæmor-

rhage may also occur. Examination of the fundi may show hæmorrhages, but papillitis is extremely uncommon.

The skin is dry, and sweating may be difficult to induce.

Response to tests for renal function.—In addition to the increased blood urea there will be a failure to concentrate urea, and usually a low diastase figure.

Course and progress.—After a period varying from a few days to many weeks, resolution may take place; the symptoms subside and dropsy disappears. A temporary polyuria often precedes the establishment of normal quantities. Albuminuria may be present for several months before finally disappearing, or it may persist indefinitely although clinically the patient is free from nephritis. In other cases its persistence indicates that the lesion is progressive and that the case is gradually assuming a chronic form. MacLean points out that this progression may take either of two directions. On the one hand, persistent urea retention, low diastase figures, and the failure to concentrate a given oral dose of urea, together with persistent cardio-vascular changes, indicate a gradual transition into a chronic interstitial type. On the other hand, persistent œdema, salt retention, a highly albuminous urine deficient in chlorides, with normal diastase reaction and normal urea concentration, are evidence of the establishment of chronic parenchymatous nephritis. The various modes of termination of an acute nephritis may be represented conveniently in the following diagram:—



Diagnosis.—Frequently the appearance of the patient is sufficiently characteristic, but, as has already been stated, œdema may be entirely absent. The diagnosis then rests on urinary examination. The significance of albuminuria must be investigated, and conditions such as functional or postural albuminuria, febrile albuminuria, and albuminuria from a failing heart must be eliminated. Hæmaturia

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must be considered in relation to chronic venous congestion and renal infarction. In cases of heart failure, dropsy with cardiac distribution may be present, but rest in bed and appropriate cardiac stimulants will usually clear up the difficulty. In renal infarction, hæmaturia will often come and go with remarkable abruptness, and sudden pain in the loin is usually a feature. The presence of epithelial and granular casts is evidence of nephritis, but hyalin casts have no such significance. Chronic parenchymatous nephritis may present similar features to the acute form both in general symptomatology and in urinary findings. It must be remembered that acute nephritis not infrequently supervenes in a chronic case. Appreciable cardio-vascular changes, persistent oedema, and albuminuric retinitis will point to the existence of an old lesion.

Prognosis.—In the majority of cases this is favourable. The cause of the nephritis, if known, is as important as its severity. Scarlatinal nephritis and the nephritis following diphtheria are sometimes quickly fatal from suppression, particularly in the young. Much depends on the duration of suppression of the urine and on the power of the kidney to respond to the treatment. The tests for renal efficiency may give valuable indication as to whether recovery is complete. The significance of these tests is referred to later (p. 357). Uræmic manifestations necessitate a guarded prognosis, but the outlook is better than in the chronic forms. Secondary inflammations such as pneumonia, pleurisy, and pericarditis are of grave omen.

Treatment.—Bearing in mind that the kidneys are acutely inflamed and their power to function is very limited, the main object in treatment will be rest for the damaged organs. This object is attained by reducing the bodily activities to a minimum and by encouraging elimination of waste products along channels other than the urinary passages. Complete rest in bed is the first essential. Cold must be carefully guarded against and the room kept at an equable temperature. The dietary is based on the knowledge that there is nitrogen and salt retention in these cases. It must be restricted in quantity and in fluid form. Milk is the best food at the outset, though it has the disadvantage of containing a considerable quantity of salt. One or two pints in the twenty-four hours is sufficient for an adult. It may be varied with gruel or thickened with arrowroot, and there is no objection

to flavouring it with tea or coffee. Barley water is useful as a bland drink. Fluids in bulk are better avoided until the urinary flow is re-established. When this takes place, sugar, cream, butter, fruit, vegetables, eggs, and fish may gradually and cautiously be given, nitrogenous foodstuffs being withheld longest. While there is hæmaturia it is better to avoid protein foods altogether and to make up a proper caloric value with carbohydrates and fats. When recovery is delayed and the disease is taking on a subacute aspect the diet should follow the lines laid down under Chronic Nephritis (p. 347).

Elimination of toxic and waste products by means of the skin and the bowel should be encouraged. Free perspiration is best obtained by means of the hot-air bath or the hot pack. These measures are especially indicated when the urine is scanty and oedema pronounced. Uræmic symptoms may likewise be relieved by their aid. The hot pack is usually the more practicable and is very efficacious, especially in children. The patient is wrapped in a blanket wrung out in hot water, covered with a dry blanket and finally a rubber sheet, and left thus for an hour. This may be repeated frequently. The hot-air bath requires caution. The body temperature should be taken frequently, and the state of the pulse and respirations noted. For the bath a temperature of 120°–140° F. is usually sufficient.

Profuse sweating is readily obtained as a rule, but if the skin does not respond the hot pack or bath may be supplemented by pilocarpine nitrate $\frac{1}{4}$ to $\frac{1}{2}$ gr., given subcutaneously. This drug should be avoided if the heart's action is weak or if pulmonary complications are present.

For the bowels nothing is better than concentrated solutions of magnesium or sodium sulphate given frequently. A drachm of either in a 1 : 2 solution every four hours may be given with the advantageous production of copious watery stools. The compound powders of jalap or elaterium are also useful. Mercury is better avoided. As to the value of diuretics there are diverse opinions. The caffeine group of drugs, diuretin and theocin, are direct renal stimulants and should be avoided in acute nephritis. The "flushing out" of the kidneys by large quantities of water is of very doubtful value. In the early stages, at any rate, the kidneys cannot excrete it, and the oedema is thereby increased.

Langdon Brown suggests that the saline

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diuretics, acetates and citrates, which act by increasing osmosis into the blood, are the only drugs likely to produce a beneficial diuresis at the expense of the oedema fluids. Citrates make the urine alkaline and therefore less irritating to the kidney.

An autogenous vaccine obtained from some focus of infection such as an inflamed tonsil may prove a useful adjunct to treatment, and is always worth a trial.

Treatment of certain other symptoms requires brief notice. Pain in the loin may be relieved by hot applications, fomentations, stupes, mustard plasters, or, better, by dry or wet cupping. Cupping, by relieving congestion in the underlying kidneys, may aid in the re-establishment of secretion.

Extreme dropsy causing embarrassment of the circulation is best dealt with by paracentesis and the insertion of Southey's tubes. Persistent vomiting should be dealt with by restricting food and giving ice to suck. The most useful drugs are the tincture of iodine given in minim doses in water every hour, dilute hydrocyanic acid, and the oxalate of cerium.

The indications for surgical treatment are briefly mentioned under Chronic Parenchymatous Nephritis (p. 347).

The secondary anaemia of acute nephritis should be met with iron. This is best withheld until convalescence is established and the digestive system is able to deal with it. It is well to begin with a mild preparation such as the double citrate of iron and ammonium. For children the syrup of the phosphate or the iodide of iron is useful. Later these preparations can be supplanted by the perchloride of iron or Blaud's pill. A change of air completes the convalescence, and preference should be given to a warm, equable climate.

The patient who has had acute nephritis should be kept under observation for several years, and frequent examinations of the urine and the renal function should be made; he should be regarded as a potential nephritic. By these means alone can the onset of chronic nephritis be diagnosed in an early and perhaps remediable stage.

The uraemia of acute nephritis is dealt with later (p. 354).

2. CHRONIC PARENCHYMATOUS NEPHRITIS (*syn.* Chronic Tubal Nephritis; Large White Kidney)

A chronic inflammation of the kidneys, affecting principally the parenchymatous ele-

ments, glomeruli and tubules, with some increase in the interstitial tissue. Clinically it is characterized by pronounced oedema with salt retention; pathologically it results in the large white kidney.

Etiology.—In some cases the disease appears to follow an attack of acute nephritis, the one gradually merging into the other in the course of some weeks. Particularly is this so in the nephritis associated with scarlet fever. In other cases the onset is more insidious and the disease makes itself evident after a considerable interval following the acute attack. Here again the specific fevers probably play an important rôle. Finally, there are many cases in which no trace of previous infection can be obtained and the etiology of which is unknown; they appear to be chronic from the outset.

Alcohol may predispose to this form of nephritis; lead and mercury are occasional antecedents. It may be associated with amyloid disease, and therefore follow in the wake of chronic tuberculosis, syphilis, or prolonged suppuration.

The disease mainly affects children and young adults.

Morbid anatomy.—To the naked eye the kidney is enlarged and may weigh 6-9 oz. The surface is smooth and usually white or yellowish-white in colour. If there has been much hæmorrhage the kidney will appear mottled. The stellate veins are conspicuous and the capsule strips easily. The cut section shows an increase in width of the cortex. The line of demarcation between cortex and medulla is well marked, the congested pyramids affording a contrast with the pallor of the cortex.

Microscopically, the most characteristic appearances are seen in the tubules. The cortical tubules are frequently dilated and the convolutions multiplied, indicating an attempt at compensatory hypertrophy. The lining cells are in varying stages of disintegration. In some places fatty changes can be demonstrated by suitable staining, whilst in others the cells have been shed and the tubules converted into scar tissue.

The glomeruli share in this process and show changes akin to those described under Acute Nephritis (p. 342). But the process has developed, the epithelium of the glomerulus and the lining membrane of the capsule having undergone proliferation, so that the capsular lumen is obliterated. At a later stage the whole glomerulus is converted into scar tissue. Inter-

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stitial changes are shown by increase in fibrous tissue throughout the kidneys with some round-celled infiltration. This fibrous tissue may be seen particularly around the capsules. The vessels exhibit endarteritis, but to nothing like the same extent as in chronic interstitial nephritis.

Symptomatology.—In the cases of this group which follow an acute attack the symptoms are in no way different from those described in the last section, but when the onset is insidious the initial symptoms may be dyspepsia, anaemia, breathlessness, and some puffiness about the eyes in the morning. Eventually dropsy becomes a prominent feature. There is general anasarca, in which the serous sacs frequently take part. The facies is often distinctive, with its pasty and pallid complexion and oedematous tissues. Wasting is considerable and becomes apparent as the dropsy subsides.

The **urine** in characteristic cases is reduced in amount to an average of half a litre daily.

granular kidney. Albuminuric retinitis may be present. Uræmia may occur, and tends to appear in chronic form. Secondary inflammation of serous membranes, such as pleurisy and pericarditis, is very prone to take place owing to the lowered resistance of the oedematous tissues. Peritonitis is a rare event; bronchitis, pneumonia, and ulcerative enterocolitis are met with. Such complications are frequently fatal.

Diagnosis.—In the majority of cases there is no difficulty, the presence of oedema with the characteristic urinary findings being usually sufficient for a diagnosis. Difficulty may arise in adjudicating the significance of albuminuria when it occurs as an isolated feature. As a rule, its characters and the previous history will show if it is to be regarded as evidence of renal disease. A careful investigation of the renal functions is essential for diagnosis and subsequent treatment. MacLean tabulates the chief points of distinction between the two main forms of chronic nephritis as follows:—

Parenchymatous Nephritis

1. Oedema present.
2. Albumin present in urine, often in very large amount.
3. Chlorides diminished or absent.
4. Urea concentration normal.
5. No urea retention in blood.
6. Cardio-vascular changes less marked.
7. Uræmia less frequent.
8. Diastase content usually normal.

Interstitial Nephritis

1. Oedema absent.
 2. Albumin present in urine, but usually small in amount.
 3. Chlorides present in normal amount.
 4. Urea concentration subnormal.
 5. Tendency to urea retention in blood.
 6. Cardio-vascular changes marked.
 7. Tendency to uræmia.
 8. Diastase content low.
-

Its specific gravity is about normal and its reaction usually acid. On standing there is an abundant deposit consisting of urates, casts of all kinds, white and red blood-cells, and amorphous debris. The albumin is large in amount, varying from 0.5 to 2 per cent., and exceptionally reaching as high as 6 per cent. Coincident with improvement and the disappearance of the dropsy there is an increase in the urinary secretion, the fluid being pale and of low specific gravity, but otherwise there is a decided retention of, and failure to excrete, chlorides and water. The kidney is able to concentrate urea in normal amount, and the diastase content is usually within normal limits.

The blood-pressure usually shows a moderate rise, and the heart may be hypertrophied and the aortic second sound accentuated, but these features are less prominent than in

Prognosis.—The outlook in chronic parenchymatous nephritis is always serious. Persistent dropsy, progressive pallor, wasting, and albuminuric retinitis are signs of ill omen. Death may occur from uræmia, secondary inflammations, or myocardial failure. Cerebral hæmorrhage is a rarer event in this type than in the chronic interstitial variety, but may occur. Of recent years the increased protein dietary suggested by Epstein has given excellent results in many cases, but it is too early to pronounce authoritatively on the results to be expected from this line of treatment. In cases in which the general nutrition is good, the oedema not excessive, the fundus healthy, and the heart unembarrassed, life may be prolonged for a number of years. Relapses, however, are the rule sooner or later, and a cure is not to be expected.

Treatment.—In the early stages, and

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especially when the urine is scanty or uræmic symptoms are imminent, treatment should follow the general lines laid down under Acute Nephritis. But the restricted diet there prescribed should only be employed to tide over a period of urgency. Loss of flesh is a characteristic feature of chronic nephritis, and the large amount of albumin lost in the urine is a drain on the general nutrition. This must be made good and the patient's well-being studied. Milk alone is an unsuitable food, and in the large amounts necessary it becomes irksome and even nauseous, while the high proportion of water and salt renders it unsuitable in dropsical cases. The supposed harmful results from a dietary containing a moderate proportion of protein may be said to be non-existent in this form of nephritis. Langdon Brown and more recently Wordley have shown that the addition of protein to the dietary does not increase the albumin output in the urine. Von Noorden found that the chronic nephritic can excrete up to 15 gm. of nitrogen daily without difficulty. This represents 94 gm. of protein, and is the maximum that should be allowed in addition to the amount lost in the urine as albumin. Langdon Brown gives the following convenient rule for calculating the last-named quantity: "When the reading of the albuminometer is 5 and the total urine 2 pints, the patient is excreting as much protein as is contained in one egg, i.e. 6 gm."

Variety in the diet is essential, and fish, poultry, and butcher's meat may be taken in moderation and within the limits prescribed above. Carbohydrates and fats may be given in normal proportion, subject to the state of the digestive organs. Meat extracts, tea, and coffee are renal irritants, and their use should be restricted. When the dropsy is excessive and persistent, a trial should be given to a so-called "salt-free" régime. Such a diet should consist of bread made without salt, sugar, any farinaceous food, butter well washed by beating it up with water, one or two eggs daily, custard, stewed or baked prunes or apples, with tea or cocoa and a little cream. Salt should be omitted in the cooking of any article. Not more than half a pint of milk must be given daily. Broths and soups must be avoided.

Unfortunately some cases remain waterlogged in spite of these restrictions, and it was for this class of case that Epstein in 1917 introduced his dietary of increased protein and

diminished fat. The rationale of this treatment lies in the lowered protein nitrogen of the blood in these cases, due to the excessive albuminuria. He attributes the oedema to the lowering of the osmotic pressure of the blood from this cause, and considers the salt and water retention as secondary to this. MacLean, however, believes that the improvement seen after this treatment is due to the non-protein nitrogen increase, especially urea, in the blood, which exerts a diuretic action on the kidneys. Be the explanation what it may, it is certain that a number of cases have shown a distinct improvement, and it should be given a trial in stubborn cases. *Epstein's diet* is as follows:—

Daily food value . . .	= 1,280–2,500 Calories.
„ protein amount . . .	= 120–240 grm.
„ fat (unavoidable) . . .	= 20–40 „
„ carbohydrates . . .	= 150–300 „

The foodstuffs used are lean veal, lean ham, white of egg, oysters, gelatin, lentils, split peas, green peas, mushrooms, rice, oatmeal, bananas, skimmed milk, coffee, tea, and cocoa. Salt is added to taste, and the quantity of fluid allowed is 1,200–1,500 c.c. Epstein particularly emphasizes the need for a prolonged course of this régime, as the improvement sets in slowly. Gradually the oedema subsides, and the benefit to general health is most evident.

Whilst a suitable diet is the basis of treatment in these cases, it may, on occasion, be necessary to relieve the dropsy by mechanical means. Diuretic drugs have proved disappointing on the whole, but when the urinary flow remains scanty a brief trial should be given to theocin-sodium acetate in 5-gr. doses three times daily. Urea itself may be tried in half-drachm doses three times a day. If MacLean's explanation of the value of Epstein's diet be correct, urea should be beneficial. The saline diuretics may also prove useful. The period of convalescence requires care. Everything must be done to avoid chills, as these cases are prone to relapses and to acute exacerbations. A warm, equable climate should be sought, the south and south-west of England being the most suitable parts of the British Isles. Tonics are necessary, especially iron. Basham's mixture, the *mistura ferri et ammonii acetatis* of the U.S. Pharmacopœia, is a useful form in which to prescribe it; the dose is a tablespoonful three times daily in plenty of water.

Surgical measures.—Decapsulation of the kidneys, Edebohls' operation, has been the subject of recent discussion. Horder considers

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that there is a type of nephritis in which—when thorough general measures have proved unavailing and a reasonable time, during which it is known that resolution not infrequently occurs, has passed—decapsulation becomes a definite indication and promises satisfactory results. This type is characterized by extensive cedema, massive albuminuria with casts in the urine, symptoms of chronic uræmia, and an absence, or the presence in slight degree only, of cardio-vascular changes. Horder and Kidd have described cases, some of which appear to have been cured, and others in which improvement has taken place at first, though they relapsed later. The operation is not a severe one, and in skilled hands takes about half an hour to perform. Improvement usually sets in at once, and within a few days there is a material lessening of cedema, albuminuria, and casts, with increase of urinary secretion. This method should be tried in cases of chronic parenchymatous nephritis which have resisted the treatment outlined above, and possibly in cases of acute nephritis with resolution delayed beyond the second month.

3. CHRONIC INTERSTITIAL NEPHRITIS

A chronic disease of the kidneys characterized by local vascular degeneration, resulting in connective-tissue overgrowth and destruction of the secretory apparatus. Cardio-vascular changes are usually pronounced.

Etiology.—Sclerosis may occur in the kidneys under the following conditions:—

- (1) Primary chronic interstitial nephritis, granular kidney, small red or contracted kidney.
- (2) The arterio-sclerotic kidney. This may occur in two forms: (a) As a sequence of general systemic arterio-sclerosis with high blood-pressure, as seen in late cases of hyperpiesis and as a result of certain chronic intoxications. (b) As a senile or decrepescient form, usually without high pressures.
- (3) The small white or so-called secondarily contracted kidney.

The relationship of arterio-sclerosis and renal disease remains obscure. In the primary granular kidney the disease begins as an arterio-sclerosis of the smallest intravisceral branches of the renal artery. From this a general systemic arterio-sclerosis may result, to support the high pressures necessary to compensate for loss of renal tissue, or may

represent the reaction of the cardio-vascular system to a poison which may arise in the kidney itself or in the suprarenal bodies.

In hyperpiesis, systemic arterio-sclerosis may occur as a secondary effect of prolonged high pressure, the kidney being one of the organs whose arteries are early affected. The senile or decrepescient kidney, according to Clifford Allbutt, represents an involutionary form. To quote his words, "There is a kidney deformed in the course of a general arterio-sclerosis not its own; and if more or less perishing by decay of its nutrient arteries, yet primarily not much amiss; a starved but not a corrupt kidney. It is but a sample of an almost universal degradation." The arterio-sclerotic kidney is functionally normal or nearly so, and is sufficiently considered in the article **ARTERIAL DEGENERATION**.

PRIMARY GRANULAR KIDNEY.—This is a disease of middle life and shows itself in the fourth and fifth decades, the majority of cases occurring in the latter, with 45 as the mean age-incidence. Heredity plays a part in its inception, some families showing the disease for three or four generations. Habitual over-eating is cited as a cause, while the anxieties of modern business and social life claim their victims. Various intoxications may precede it. Of these gout, syphilis, and to a lesser extent diabetes are prominent. Alcohol and lead among chemical poisons are undoubtedly predisposing factors. A very rare form of granular kidney is seen occasionally in infants. The symptoms originate either from birth or after four or five years of life. Cases have been described by Morley Fletcher, Parsons, Miller, and others. They exhibit varying degrees of infantilism (q.v.).

Morbid anatomy.—The kidneys show great variation in size. Typically they are much smaller than normal and reddish in colour. The weight ranges from 6 to 1½ oz. In the early stages naked-eye changes are slight, the cortex and medulla are in normal proportion, but the vessels are unduly prominent. Later the surface is granular and the capsule is adherent to the renal tissue. Small cysts may be evident; the cortex is greatly diminished in width, and vascular changes are pronounced.

Microscopically, the interlobular and afferent arterioles show great thickening of their walls and in some places obliteration of their lumens. In these areas the glomeruli are converted into masses of hyalin fibrous material. There is

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a wide destruction of tubules. A very characteristic feature of the lesions is the patchy distribution, areas of fibrosis alternating with seemingly healthy areas.

The arterio-sclerotic kidney associated with hyperpiesis is not necessarily a contracted kidney. It may, indeed, be heavier than normal, is hard to the touch, and has a red beefy appearance. The surface may be smooth or somewhat warty, and there is usually little adherence between the capsule and renal tissue. There is general systemic arterio-sclerosis, and cardiac hypertrophy may be enormous.

The senile or decreascent kidney is smaller and softer than the primary granular form and shows greater deformity. The areas of fibrosis are larger, and cause depressed scars on the surface resembling old infarcts. Associated with this condition are evidences of general arterio-sclerosis, though usually without cardiac hypertrophy.

Symptomatology.—The onset is usually very insidious. In some cases the first manifestations are renal; in others they are referable to cardio-vascular changes. In not a few, sudden uræmic symptoms or a cerebral hæmorrhage may be the initial evidence of disease. Of the earliest symptoms, mention may be made of disordered digestion, headache, giddiness, lassitude, failing vision, shortness of breath, nocturnal frequency of micturition, loss of flesh, and progressive anæmia.

The urine.—Polyuria is common, and the night urine frequently exceeds the daily quantity. The colour is pale and the specific gravity low, varying between 1005 and 1012. The deposit is scanty, but usually contains a few hyalin and granular casts. Blood is rare, except as a result of heart failure or of an acute exacerbation; albuminuria is by no means constant, and the amount usually no more than a light cloud on boiling. Albumin-free intervals are characteristic of the disease.

Cardio-vascular system.—The pulse is slow and sustained. There is a high blood-pressure picture, systolic readings being from 200–300 mm. Hg, and diastolic 120–140 mm. Hg. A high pulse-pressure and ultimately cardiac hypertrophy are the result. The impulse is forcible, the first sound accentuated and often reduplicated. The aortic second sound is high-pitched. With the onset of failure the pulse-pressure falls, though the general level may remain high. The heart quickens and gallop rhythm may occur, together with an apical systolic bruit indicative of relative mitral

insufficiency. Finally, œdema of cardiac distribution may show itself.

Respiratory system.—Dyspnoea, especially at night, is apt to occur apart from pulmonary complications; it has a grave significance. It may be asthmatic in character and indicative of uræmia, or it may show the Cheyne-Stokes periodicity, especially towards the termination.

Winter cough with bronchitis and emphysema are common accompaniments. Laryngeal œdema may occur with startling and fatal suddenness, as also may pleural and pericardial effusions. Pleurisy and pneumonia are met with as complications.

Nervous system.—Apart from uræmic manifestations, cerebral hæmorrhage may occur as a direct result of the high blood-pressure and general vascular disease.

Headache is a very common complaint and often associated with rising blood-pressure.

Special sense-organs.—Conjunctival hæmorrhages may occur. The ocular fundi may show the changes associated with high blood-pressure—uneven calibre and undue tortuosity of the vessels, loss of translucency, the silver-wire arteries, the compressed veins, and flame-shaped hæmorrhages. Albuminuric retinitis is very common in this form of nephritis. Epistaxis may occur and prove very intractable, though it may bring decided relief to headache and perhaps be the means of averting a cerebral apoplexy. Recurrent epistaxis in those of middle and advancing years, in the absence of local disease, should suggest chronic nephritis.

Response to tests for function.—This form of nephritis is associated with nitrogen retention. The blood-urea is therefore high and in severe cases may reach the figure of 300 mg. per 100 c.c. of blood. The urinary urea, though low in any given specimen, may show a normal output in twenty-four hours. Its excretion is so largely dependent on the diet that nothing is to be learnt from its estimation unless the daily intake is accurately measured. This is usually impracticable. MacLean's urea concentration test, however, gives information of much value, since in the primary granular kidney the power to concentrate is wanting. The diastase content is usually subnormal, but there is no retention of water and salt.

Diagnosis.—All too frequently the diagnosis is made by the pathologist at autopsy. The insidious onset, often latent symptoms, and widespread cardio-vascular disease mask the renal origin. In many cases the diagnosis is

not in doubt, particularly in younger subjects, in whom vascular changes are not so obtrusive. Careful and frequent examination of the urine and the measurement of the renal response to function-testing are essential for the forming of a correct opinion. Blood-pressure readings and ophthalmoscopic examination are of the greatest value. The personal history and habits of the patient may have an important bearing. There is the question of an hereditary taint and the possibility of antecedents such as gout, syphilis, chronic alcoholism, and perhaps lead intoxication.

In the comatose patient granular kidney is always a possibility. The coma may be due to a cerebral hæmorrhage or to uræmia. In either case urinary examination is called for.

Primary hypertension or hyperpiësis (Allbutt) may present symptoms similar to those of primary granular kidney. The two conditions have certain etiological factors in common, in that both are met with amongst men who lead active business lives and who "do themselves well." Hyperpiësis may be distinguished by its somewhat earlier age-incidence and by the lack of evidence of renal involvement, either from urinary examination or from investigation of function. Uræmia is not a complication of hyperpiësis, but cerebral hæmorrhage may occur.

Granular kidney may be distinguished from the *senile kidney* by its earlier incidence, the latter being an accompaniment of senile decay. In the senile form there is usually no polyuria and the specific gravity may be normal. Albuminuria there may be, and a few casts as well. The vascular changes are those met with in later life. The blood-pressure is not necessarily high, nor is cardiac hypertrophy common.

Prognosis.—Primary granular kidney is a progressive disease to which the term "cure" is inapplicable. Its progress can, however, be retarded and life prolonged for several years by removing any source of aggravation and attending to the diet and mode of life. The duration of the disease shows a mean figure of fifteen years. Allbutt quotes figures giving the average age at death as 56. Albuminuria, casts, and high blood-pressure are not in themselves necessarily dangerous, but they indicate the existence of conditions which may rapidly become so. The points of prognostic value in blood-pressure readings are: (1) A rising systolic pressure is a bad sign, and not infrequently precedes the onset of convulsions. (2) A persistent high diastolic pressure, e.g.

150 mm. Hg or more, predisposes to cerebral hæmorrhage. (3) When the systolic and diastolic pressures approach one another, heart failure is imminent, be the pressure picture high or low (Warfield). Uræmic manifestations, albuminuric retinitis, and periodic dyspnœa have each a grave significance. The blood-urea is usually high in proportion to the severity of the case, and any figures over 100 mg. per 100 c.c. of blood warrant a grave prognosis. It must not be forgotten, however, that the blood-urea may be within normal limits.

Treatment.—It is rare for this disease to be detected in the earlier stages, and Osler has well said that the man of middle years whose trace of albumin is discovered is to be congratulated. When the disease is recognized, a régime must be established forthwith which will throw the minimum of strain on heart, arteries, and kidneys. Any possible source of irritation must be removed and bodily and mental activities reduced. Cold must be guarded against, and care devoted to dietetic considerations.

The indications for special dieting are not so clear in this disease as in chronic parenchymatous nephritis. The chief difficulty here appears to be in the elimination of non-protein nitrogenous substances, of which urea forms the largest part. A dietary low in protein would thus be indicated on theoretical grounds. Practically, protein in moderation does not appear injurious, and Langdon Brown suggests that the daily quantity should be between 60 and 90 gm. A pint of milk, 2 oz. of meat, and an egg contain 72 gm. of protein and form a rough guide.

Carbohydrates and fats may be given in normal proportions. In severe cases in which there is much nitrogen retention an occasional meatless day may be of distinct value, but it is difficult to persuade some patients to submit to it. Such meatless days should be supplemented by taking an extra amount of carbohydrate and fat, the latter being ingested in the form of butter, cod-liver oil, etc., and the carbohydrates as cereals, bread, potatoes, green vegetables, and fruit. Alcohol in any form is forbidden. Meat extracts and offal are renal irritants and should be avoided. Tea, coffee, and cocoa contain purin derivatives and should be used sparingly. Some restriction of fluids is advisable in order to spare the embarrassed circulation as much as possible, and for the same reason salt should be taken in reduced amount.

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Special treatment may be required for cardiovascular symptoms, chief among which are high arterial tension and heart failure. High blood-pressure, *per se*, is not necessarily harmful. It is probably a compensatory mechanism and its reduction may do harm. When, however, the pressure is rising and is evidently associated with symptoms such as headache and breathlessness, indications for its relief are present. Rest in bed and a light dry diet should be tried, and the bowels kept well open by saline aperients. These measures may be supplemented by a daily hot-air bath. Drugs for the relief of high tension are frequently disappointing owing to their transient effect. Warfield recommends a mixture of tincture of aconite and spiritus etheris nitrosi. The best are erythrol tetranitrate and mannitol nitrite. They are conveniently combined in the tabellæ sodii nitratis co. (Oliver). If the above measures fail or if symptoms become urgent, venesection should be resorted to, and a pint to a pint and a half of blood removed. Occasionally spontaneous hæmorrhage such as epistaxis may bring about relief. Lumbar puncture is often useful, and may be performed without hesitation.

Heart failure, as evidenced by falling pressure, increase in pulse-rate, cardiac dilatation, gallop rhythm, and œdema, should be met by cardiac tonics such as digitalis and caffeine in full doses.

Anæmia, often a prominent feature, requires iron. The perchloride of iron and Basham's mixture (mist. ferri et ammonii acetatis, U.S.P.) are the most useful preparations.

Climatic considerations are important, and a warm, equable climate should be chosen for winter quarters. For the well-to-do, Southern Europe, North Africa, and Egypt offer the ideal conditions.

CONTRACTED WHITE KIDNEY.—A rare form of chronic nephritis, of unknown origin, occurring in young adults. The symptomatology resembles that of the chronic interstitial nephritis of later life. Its pathological status is obscure.

Etiology.—There are two opinions as to the cause of the small white kidney. It may represent a late stage of the large white, in which case it would be a mixed type, intermediate between parenchymatous and interstitial nephritis. A few cases possibly own such an explanation, but the great majority of patients give no previous history of scarlet

fever or acute nephritis. Furthermore, clinically there is no resemblance to the large white form, but a close relationship to the small red kidney. Rose Bradford was the first to draw attention to this group of cases, which he considers to have evolved along lines entirely separate from the parenchymatous form. He believes this to be a disease *sui generis*, and the description that follows is based upon his writings.

Age-incidence.—The disease occurs during the second, third, and fourth decades of life, but most commonly in the third.

Morbid anatomy.—The kidneys are much reduced in size and weigh 1–3 oz. The surface is granular, but as a rule the capsule strips easily. On section the cortex is much diminished in size, is pale in colour, and blends indistinguishably with the medulla. Microscopically, the glomeruli are small, and Bowman's capsule is greatly thickened, resulting in compression atrophy of the glomerular tuft. The tubules show much loss of epithelium and its place is taken by fibrous tissue. In other parts, where the epithelium is intact, it may show different stages of degeneration. Dilated tubules lined with flattened epithelium are also seen. The vessels show pronounced periarteritis and endarteritis. The atrophy appears to be due to a loss of tubular elements. Histologically the resemblance to the red granular kidney is close, but tubular destruction is usually more evident in the small white kidney.

Symptomatology.—The onset is usually very insidious, and the disease may not be recognized until ushered in by acute uræmic symptoms. Nutritional disturbances are prominent, wasting and anæmia being features which may be so evident as to suggest malignant cachexia. Polyuria and consequent thirst are frequent symptoms. The urine is pale in colour and of low specific gravity; it contains a moderate amount of albumin. Cardio-vascular changes are common; the blood-pressure is high and the heart hypertrophied. Præcordial pain and palpitation are complained of, while headache is often severe.

Albuminuric retinitis is more common in this than in any other form of nephritis. Œdema is not met with, except as a result of heart failure.

Uræmic symptoms are usually of the acute type, and may be the first evidence of ill-health. Sudden uræmic dyspnoea may herald

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the fatal termination, and convulsions are sometimes met with.

Diagnosis is based on the age of the patient, the characters of the urine, and the presence of cardio-vascular disease. Œdema is usually absent, while pallor and wasting are often prominent. The response to tests for renal function is not known. The appended table represents the main clinical differences between the large white and the small white kidney.

Small White Kidney

1. Dropsy very uncommon.
2. Cardio-vascular changes prominent.
3. Nutritional disturbances pronounced.
4. A disease of young adults.
5. Albuminuric retinitis common.
6. Latent course, with rapid terminal uræmia.

Large White Kidney

1. Dropsy common.
2. Cardio-vascular changes not so well marked.
3. Nutritional disturbances not so evident.
4. Not confined to the young.
5. Albuminuric retinitis seen occasionally.
6. Course usually not latent, and uræmia chronic in type.

Prognosis.—The disease is invariably and rapidly fatal, death in the large proportion of cases occurring within a few weeks or months of its recognition.

Treatment.—Very little can be done. Generally speaking, the treatment of primary interstitial nephritis should be followed. Uræmic symptoms necessitate vigorous measures.

4. WAR NEPHRITIS (TRENCH NEPHRITIS)

An acute nephritis of unknown cause occurring among troops on active service.

The occurrence of acute nephritis among soldiers in the field was a striking feature of the medical history of the European War, all the belligerent armies being affected to a greater or less extent. In previous campaigns, with the exception of the American Civil War, nephritis appears to have been uncommon.

Etiology.—In spite of research in many directions, the cause of trench nephritis remains uncertain. MacLean and de Wesselow, investigating the incidence of albuminuria among soldiers in training, have shown that it played no part, nor was pre-existing nephritis a factor. Exposure to damp and cold doubtless had its effect, but the incidence among those in exposed areas was often less than among those under better conditions. Again, a considerable number of cases occurred among men who had recently arrived in the front area. The following suggestions as to causation have been made :—

(1) Poisoning by metals, especially lead, in water or derived from tins containing food.

(2) Unsuitable diet, particularly an excess of protein.

(3) Deficiency in vitamins.

(4) Infection through the air-passages. The frequent association of bronchitis made this a possible channel of entry.

(5) Infection by a filter-passing organism.

(6) Infection by means of lice-bites.

All but the last three of these factors have

been amply disproved. There is evidence to support the theory of lice-borne infection, but not sufficient to establish it. The nature of the disease is strongly suggestive of an infective origin. Scarlet fever played no part, nor could a relationship to trench fever be established. Bacteriological investigations of the urine, blood, throat, and faeces proved negative.

Prevalence.—The great majority of cases occurred among troops in the forward area, but a few instances were noted among nursing sisters and hospital orderlies at the base. Officers seem to have been rather less liable to the disease than the rank and file.

The curve of incidence was in inverse proportion to the temperature, the winter months showing the largest percentage. The disease reached its maximum in December, 1916.

Morbid anatomy.—Shaw Dunn and McNeen described in detail the appearances seen in 35 autopsies, conspicuous uniformity being observed in the series. Briefly, the changes described by them were these: The kidneys showed little change to the naked eye. There was no material alteration in size, and the capsule stripped readily. On section the cortex was pale and the pyramids were congested, the glomeruli being prominent as pale translucent globules. Microscopically, the lesion was essentially a glomerular nephritis, the glomeruli being more cellular than normal and containing little or no blood. The ostia of the convoluted tubules were plugged in many instances by herniations of the glomerular vessels. The

capillaries showed no hyalin thrombi, but often contained polymorphonuclear leucocytes and lymphocytes. The capsules rarely showed any epithelial proliferation; fibrinous exudate and hemorrhages were rare. The tubular lesions were slight, various stages of degeneration being observed occasionally. Hemorrhage into the tubules was not uncommon, and casts of epithelial and hyalin type were seen.

There were no characteristic changes in the interstitial tissue.

Changes in other organs were sufficiently striking to merit brief description. The lungs in the majority of cases were voluminous, heavy with oedema, and showed extensive emphysema. Much frothy fluid exuded from the cut surface, and diffuse subpleural hemorrhages were present. Microscopically, oedema and lobular consolidation were noted, and, in addition, characteristic changes in the terminal bronchioles and infundibula. The walls of these cavities were swollen with oedema and denuded of epithelium, its place being taken by a layer of dense fibrinous material.

These findings led Dunn and McNee to formulate the view mentioned above, that a labile irritant, either inhaled or excreted from the blood, was the cause of the condition.

The brain showed in some cases minute hemorrhages in the white matter, especially of the frontal and occipital lobes.

Symptomatology.—The onset was either acute or insidious, the majority of cases falling into the latter group. Generally speaking, the symptoms resembled closely those of the nephritis of civil practice, but were of a greater intensity. Severe headache, dyspnoea, and oedema were marked features. Abercrombie recognized two distinct types of dyspnoea—one with cyanosis, in which pulmonary complications were present, and the other without cyanosis. Keith and Thompson have shown that in a few cases the latter type depended on an acidosis.

Pulmonary complications—bronchitis, broncho-pneumonia, emphysema, and oedema—were not uncommon.

Convulsions were present in about 4 per cent. of cases, their early occurrence being especially noteworthy.

The distribution of oedema, the urinary findings, and cardio-vascular phenomena differed in no way from their parallels in the nephritis of civilian practice. The blood-pressure usually showed a moderate rise to 160 mm. Hg or thereabouts. Retinal changes were common

and were dependent generally on the degree of vascular change, the usual findings being hemorrhages, retinal oedema, and exudation.

Course and progress.—The great majority of cases made a good recovery so far as immediate symptoms were concerned. The mortality was 1 per cent. in the acute stages and 2-5 per cent. in the later stages. Recovery appeared to be complete in about 60 per cent., and in 40 per cent. more or less permanent renal damage remained. But it is too early to say if the term recovery will be justified by subsequent events.

The evidences of recovery usually appeared between the fourth and fourteenth day from the onset, copious diuresis and a rapid disappearance of oedema taking place. In some cases resolution was delayed, the oliguria and oedema persisting; in others, even though resolution appeared to be satisfactory, investigation of the renal function showed evidence of latent disease. MacLean found that these two types progressed along different lines. In the one, the persistence of oedema and oliguria betrayed a transition to the chronic parenchymatous or hydræmic form; and in the other, nitrogen retention, a failure to concentrate urea by the special test, and a low diastatic activity indicated the onset of chronic interstitial nephritis, the azotæmic variety.

Prognosis.—Prognosis thus depended on early resolution, and subsequent application of functional tests indicated its completeness.

Treatment.—This follows the lines laid down for acute nephritis. Venesection, especially, proved of great value in cases with rising blood-pressure and for convulsions.

5. RENAL SYPHILIS

The kidneys are liable to damage from the syphilitic virus in the early and in the late stages of the disease, but it is only to the former that the term nephritis can be properly applied. Following Dieulafoy, we have—

- (1) Syphilitic nephritis.
- (2) Tertiary renal syphilis.

(1) **SYPHILITIC NEPHRITIS.**—Coincident with the evidences of a generalized syphilitic infection there may be an albuminuria without evidence of nephritis. It appears within a few months of infection, and is generally a transitory phenomenon. The amount of albumin passed varies from 3 to 5 gr. daily; casts are not found, and there is no derangement of function. Such cases usually

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show an uneventful recovery under anti-syphilitic measures.

A true nephritis, however, occurs in about 3-8 per cent. of cases in the secondary stage. It usually shows itself in the second or third month following infection, with the other manifestations of this period—the rash, condylomata on the mucous membranes, glandular enlargement, and sore throat.

Symptomatology.—The main features of syphilitic nephritis are sudden onset, widespread œdema, and pronounced albuminuria. In other respects it conforms to the ordinary type. The œdema shows a widespread anasarca, with frequent involvement of serous membranes, especially the pleuræ and peritoneum. The urine contains a very high percentage of albumin, 20-30 grm. being passed in the twenty-four hours. Blood and casts may be present.

Diagnosis.—The occurrence of rapid œdema and abundance of albumin in a case of nephritis suggest a syphilitic origin, and a search should be made for other evidences of secondary syphilis. Of these, the presence of a rash is most helpful. Where doubt exists the Wassermann reaction should be employed.

Course and progress.—Recovery is usual if the case is vigorously treated, but death may occur from widespread dropsy, heart failure, or uræmia. As in other forms of nephritis, the dropsy may persist and the case take on the characters of the large white kidney.

In other cases clinical recovery appears to be complete, but later an acute exacerbation occurs or the features of chronic nephritis may very gradually appear.

Treatment.—In addition to the general principles of treatment and diet laid down under Acute Nephritis, antisyphilitic measures are essential. Mercury is not contraindicated in the nephritis of secondary syphilis. It should invariably be prescribed, but in moderate doses and with a watchful eye for evidences of mercurialism. There are diverse opinions as to the wisdom of giving salvarsan and its modifications in cases of syphilitic nephritis. Most authorities advocate its use, but the initial doses must be small in order to avoid toxic effects from deficient elimination. An adequate initial dose of novarsenobillon or neokharsivan is 0.2 grm.

With the reservations mentioned, the routine treatment for syphilis should be followed.

If the nephritis becomes chronic, the diet

recommended for chronic parenchymatous nephritis is indicated.

2. **TERTIARY SYPHILIS OF THE KIDNEY.**—This manifests itself as—

(a) *Chronic interstitial nephritis.*—The syphilitic toxin may be an antecedent to this form of nephritis by giving rise to arterial disease. There is patchy fibrosis in the renal cortex.

(b) *Gummata.*—These occur occasionally. Usually the tumours are small, multiple, and undiagnosed during life. Very rarely a palpable renal tumour may prove to be a gumma. The point is worth keeping in mind, since such a tumour would be amenable to antisyphilitic measures.

(c) *Amyloid disease.*—This is considered under AMYLOID DISEASE.

URÆMIA

The name given to a symptom-complex occurring in nephritis, and in other conditions associated with anuria.

Pathology.—The various theories put forward to explain uræmia may be divided into chemical and mechanical. The main *chemical* theories are—

(1) That substances normally excreted by the kidneys are retained in the body. Chief among such bodies are urea and potassium salts. Blood-urea figures do not support this view; uræmia may be associated either with an increase or a diminution. Bouchard's theory of potassium retention is discredited, since the salts of urine are less toxic on injection into animals than the whole contents.

(2) That intermediate products of protein metabolism are formed in excess, notably ammonium carbonate. This was observed first in dogs with an Eck fistula, and was thought to account for the symptoms they presented. It has not been found in the blood of uræmic persons.

(3) That the kidneys normally produce an internal secretion whose failure may result in uræmia. Rose Bradford has shown that this secretion has an inhibitory effect on protein metabolism, but that when the kidney substance is reduced to a quarter of its original amount this effect breaks down, and the blood and tissues are flooded with protein metabolites. As mentioned above, high blood-urea figures are not a necessary association of uræmia; and, further, the kidneys may present to naked-eye and microscopical examination an appearance not far removed from normal.

4. That the damaged kidneys give rise to certain bodies known as nephrolyns, which exert a specifically destructive action on renal tissue. Very advanced renal lesions may be present, however, in the absence of uræmia.

5. That uræmia is the result of an acidosis which has been shown to exist in a large number of cases. Certain symptoms, notably dyspnoea, are in all probability due to this factor, but it is an association rather than a cause of uræmia.

On *mechanical* grounds uræmia is attributed to oedema of the brain and spinal cord. The wet brain is a not uncommon finding at autopsy, and may perhaps give rise to certain of the uræmic phenomena.

To sum up, none of these theories has met with general acceptance, and it is probable that many factors are at work to produce the symptom-complex. These factors in all likelihood vary with the type of the underlying renal lesion and the form of uræmia associated with it.

Symptomatology.—Uræmia is met with in three main forms: (1) *Acute* uræmia. This occurs in any form of nephritis, but especially in the small white kidney. Uræmic manifestations may be the initial evidences of ill-health, and convulsions or urgent dyspnoea are usually the first symptoms. (2) *Chronic* uræmia is met with in chronic parenchymatous and chronic interstitial nephritis. Its course may be long drawn out, headache and gastro-intestinal symptoms preceding terminal events by several weeks. (3) *Latent* uræmia is the term applied to those events which succeed complete arrest of the renal function, such as occurs in obstructive anuria. The characteristic symptoms of uræmia are usually absent. In these cases the patient remains well until about the seventh day, after which death may occur suddenly. The symptoms are slight, but vomiting, dyspnoea, and twitching of muscles may be present.

Cerebral symptoms.—*Convulsions* are common, and frequently usher in the uræmic attack. They vary from a localized twitching of the face or limb muscles to a generalized convulsion indistinguishable from major epilepsy. The attacks are prone to recur at variable intervals. In some cases these are so short as to resemble the condition of status epilepticus. The sensory auras of epilepsy are usually absent. Death may occur during the first or any subsequent convulsion. *Paralyses* of monoplegic or hemiplegic distribution are occasional signs, and may be associated with

aphasia. They may follow a convulsion. The organic basis of these paralytic phenomena is not fully understood. Oedema of the brain is the only lesion which is at all constant at autopsy. A feature of the uræmic palsies is their transitory nature, complete recovery being the rule. *Coma* is very common. The onset may be gradual as in chronic uræmia, when it is preceded by headache, mental apathy, drowsiness, and vomiting, or it may be sudden and result from a convulsion. The coma may resemble that which accompanies the typhoid state. The state of the pupils is not characteristic. *Delirium* may occur in any form and at any stage of the disease, and may lead to *mania* of the most violent character. Delusions (the *folie brightique*) are not uncommon and generally take the form of persecution. *Headache* is a very constant symptom but has no special characteristics or distribution; occasionally it is migrainous in character. *Sudden blindness* may occur in uræmia, and be quite independent of any albuminuric retinitis or papillitis which may be present; in fact, examination of the fundus is usually negative. Uræmic amaurosis is characterized by its sudden onset and transient nature. It may occur as a sequel to a convulsion or be an isolated phenomenon. Its duration varies from a few hours to a few days. The lesion is cortical, and the pupillary reactions to light are normal. Partial or complete *deafness* is rare, but tinnitus is not uncommon.

Gastro-intestinal symptoms.—Loss of appetite and nausea are usual early symptoms, and vomiting of cerebral type may occur. The tongue is generally furred, and may present the dry, brown, and cracked appearance met with in the typhoid state. Stomatitis has been described. Diarrhoea occurs sometimes as a terminal event and may be associated with ulcerative colitis. Hiccough is occasionally a persistent and intractable symptom, and its significance is grave.

Cardio-vascular symptoms.—The condition of the heart and blood-vessels is dependent on the type of nephritis present. The blood-pressure varies considerably. Generally speaking, the outlook is better if the pressure is raised than if it is subnormal. A rising blood-pressure is occasionally a precursor of uræmic symptoms, especially of the convulsive type. The pulse is usually slow and of good volume, but tends to become rapid and full when convulsions supervene.

Uræmic dyspnoea.—Three types are met

NEPHRITIS

with: (1) Continuous dyspnoea of laboured and noisy character. (2) Asthmatic breathing; this is usually associated with high blood-pressure, and occurs most frequently at night. (3) Cheyne-Stokes respiration is encountered in the chronic forms of uræmia, and may show itself some weeks or even months before the fatal termination.

The urine and the blood.—The chemical constitution and amount of the *urine* will depend on the form of nephritis which is present. Commonly the output is scanty, and there may even be suppression. Nevertheless, uræmia can occur when the output is normal in quantity—a point which deserves emphasis. The amount of urea varies, but, speaking generally, there is a diminution in nitrogenous substances. The albumin is very variable in amount; on rare occasions it may be absent altogether. Blood and casts are frequent findings. The *blood* usually but not invariably shows a raised urea content; it may amount to as much as 300 mg. per 100 c.c. of blood, in which case a fatal termination is inevitable. On the other hand, cases of uræmia occur with the blood-urea within normal limits. An acidosis is frequently present, and may be measured by the amount of sodium bicarbonate required to make the urine alkaline. For details of this test and its clinical application the reader is referred to the monograph on Acidosis by A. W. Sellards.

The temperature is variable. In the acute form it may be raised; in the chronic it is more usually subnormal. Any distinct departure from the normal is an unfavourable sign.

Cutaneous symptoms.—Itching may be a prominent and troublesome feature, and in its presence a very grave prognosis should be given. A characteristic eruption has been described, maculo-papular in character, with an initial distribution over the extensor surfaces of the limbs. Later it tends to spread over the body, and the lesions may coalesce. Desquamation may occur, or the rash may take on eczematoid characters.

Diagnosis.—The occurrence of any of the foregoing symptoms in a case of nephritis will arouse suspicion. The diagnosis in a comatose patient may, however, be a matter of great difficulty, and it will be necessary to exclude such conditions as cerebral hæmorrhage, fracture of the basis cranii, postepileptic coma, acute alcoholism, general paralysis, opium poisoning, and diabetic coma. The odour of the breath may give useful help; in uræmia it has, not infrequently, a urinous character.

A routine examination of the nervous system should be carried out in all cases, particular attention being paid to the size, equality, and light-reaction of the pupils, the condition of the fundi, the occurrence and distribution of palsies, the spasticity or flaccidity of the limbs, and the character of the tendon-reflexes and plantar response. In uræmia the pupils are usually equal, of varying size, and reacting normally to light. The fundi may show albuminuric retinitis. Palsies may occur, usually of hemiplegic distribution, which are indistinguishable from those of gross intracranial diseases. Generally, however, they are transient. The tendon-reflexes are variable but commonly are exaggerated, and the plantar response may be of extensor type, particularly after convulsions.

A lumbar puncture will often give valuable assistance. In uræmia the cerebro-spinal fluid is generally under increased pressure, and may contain from 0.1 to 0.6 per cent. urea, whereas the normal figure is about 0.05 per cent. The presence of altered blood will indicate intracranial hæmorrhage. A negative Wassermann reaction is evidence against syphilis of the nervous system. Convulsions have to be distinguished from those due to epilepsy and gross intracranial disease.

Examination of the urine is of primary importance in all cases, and a specimen should be obtained by catheter if necessary. Evidence of renal disease should be sought for, and the possibility of a cerebral hæmorrhage occurring in a case of chronic interstitial nephritis borne in mind. In the chronic form of uræmia a typhoid state is not uncommon, and a Widal test may be necessary to exclude enteric-group disease. Myxœdema in its late stages may closely simulate chronic uræmia, and, indeed, nephritis is a termination not infrequently met with in such cases.

Estimation of renal function in uræmia is probably of little assistance beyond indicating the type of nephritis present. The blood-urea may be very greatly increased, but, on the other hand, uræmia can occur when it is subnormal.

The presence of glycosuria with acetone bodies in the urine, together with the typical air-hunger, will point to diabetic coma.

Prognosis is usually bad; it must always be guarded. Complete recovery may take place, particularly in the uræmia associated with acute nephritis, and this in spite of such symptoms as convulsions and amaurosis. In chronic interstitial nephritis uræmic symptoms

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have always a serious import, and a fatal recurrence at no distant date can be predicted. Very high concentration of blood-urea (e.g. over 100 mg. per 100 c.c. of blood), a marked failure to respond to MacLean's urea-concentration test, and a low diastase figure, all indicative of serious renal inadequacy, are ominous signs. The urea percentage in the cerebro-spinal fluid approximates to that in the blood. Its estimation, however, is a much simpler matter, and can be carried out by the methods of Gerrard or Doremus, using 5 c.c. of the fluid. The results appear to show that with between 0.1 and 0.2 per cent. urea many cases prove fatal, whilst with over 0.2 per cent. the outlook is invariably hopeless. Cheyne-Stokes respiration has an equally ominous significance. The immediate prognosis depends on the power of the kidney to function and on the response to treatment.

Treatment.—Establishment of the renal function and elimination of poisonous products constitute the objectives in treatment. If the urine be scanty or suppressed, counter-irritation over the loins by dry cupping is useful. Occasionally the injection of hot saline into the rectum produces the necessary diuresis. Diaphoresis is important and is most quickly obtained by means of the hot-air bath or hot pack. The injection of pilocarpine nitrate, $\frac{1}{2}$ to $\frac{3}{4}$ gr., is a valuable adjunct, the precautions referred to under the treatment of acute nephritis being borne in mind. Active purgation must be carried out, concentrated solutions of magnesium or sodium sulphate being the most efficacious means. In the comatose patient 1 min. of croton oil in butter is indicated. For convulsions, venesection promptly performed is often of great value. Special indications for its use are a rising blood-pressure, exaggeration of tendon-reflexes, and muscular twitching. Such signs indicate the approach of convulsions, and a timely venesection of 15–30 oz. of blood appears to prevent their occurrence in some cases. Lumbar puncture is of value also in such cases, and may conveniently be combined with venesection under a light anaesthesia.

Drugs for the control of convulsions are chloral hydrate, 10–20 gr., and the bromides, $\frac{1}{2}$ –1 dr., either by mouth or by rectum; in very severe cases morphia should be given without hesitation. For the headache the coal-tar preparations, phenacetin, antifebrin, phenazone, and aspirin, are useful; whilst in cases in which the headache is associated with

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high blood-pressure nitro-glycerine, $\frac{1}{10}$ gr., or one of the nitrates, of which erythrol tetra-nitrate and mannitol nitrate are the best, may be given three times a day. For immediate relief the inhalation of amyl nitrite is often of service. Uræmic dyspnoea should be treated with continuous inhalations of oxygen, and with full doses of sodium bicarbonate to correct any acidosis that may be present.

For the restlessness, insomnia, and delirium hyoscine hydrobromide is the most reliable remedy we possess. It may be given in doses of $\frac{1}{150}$ gr. for a woman and $\frac{1}{100}$ gr. for a man, and be repeated if necessary. Cautiously administered, its use is unattended with danger, and will often succeed where morphia fails.

T. L. HARDY.

NEPHROLITHIASIS (see URINARY CALCULI).

NEPHROPTOSIS (see VISCEROPTOSIS).

NERVES, CRANIAL, LESIONS OF (see NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF).

NERVES, INJURIES TO (see HEAD INJURIES; SPINAL NERVES, LESIONS OF).

NERVES, SPINAL, LESIONS OF (see SPINAL NERVES, LESIONS OF).

NERVES, TUMOURS OF (see NEUROMA).

NERVOUS DYSPEPSIA (see STOMACH, FUNCTIONAL DISORDERS OF).

NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF.—Any attempt to enumerate the symptoms of local lesions of the central nervous system involves in most cases the artificial construction of a syndrome consisting only of such symptoms as are directly referable to the structures included in the area of the lesion, all symptoms that arise from secondary involvement of other nervous structures, either by their contiguity to the lesions or by indirect disturbance of their functions, being disregarded. A local lesion causing only symptoms due to the structure immediately involved is, therefore, a clinical abstraction in the great majority of cases, and its description is merely useful for purposes of reference.

Local lesions of the *spinal cord* are dealt with in a separate article (see SPINAL CORD, LOCAL LESIONS OF).

Local lesions of the brain-stem.—The

NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF

brain-stem includes the medulla, the pons, and the cerebral peduncles, together with the corpora quadrigemina. In the ventral area of

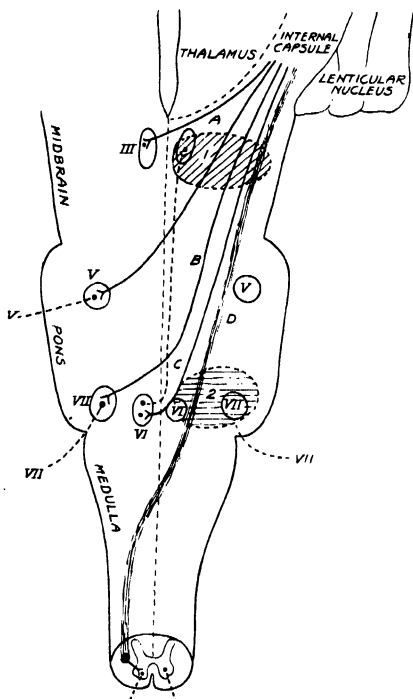


Fig. 54.—Diagram of alternating paralysis.

A, Cortical fibres to the oculo-motor nucleus; B, cortical fibres to the facial nucleus; C, cortical fibres to the 6th nucleus; D, cortical fibres to the spinal cord. (For lesions [shaded] see text.)

the brain-stem are crowded together all the important nerve tracts connecting the mid- and fore-brain with the spinal cord, whilst in its dorsal part lie the nuclei of all the cranial nerves, except the first two pairs. While, therefore, a very small lesion may give rise to a multiplicity of symptoms by causing an interruption of several tracts, its level can often be localized with great accuracy if the contiguous cranial-nerve nuclei are affected, or if the cortico-bulbar tracts going to them are interrupted. In the first case there will be an atrophic paralysis of the muscles supplied by the cranial nerve; and in the second

case, a distant paralysis without atrophy. The motor tract contains, in addition to fibres for the motor nuclei of the spinal cord, fibres that pass to the motor cranial nerve nuclei of the opposite side, and these decussate at different levels. A glance at the diagram (Fig. 54) shows that a lesion, 1, situated at the level of the oculo-motor nuclei, will cause paralysis of the 3rd nerve on the same side and a paralysis of the facial and skeletal musculature on the opposite side, since the fibres to the face and body undergo decussation below the level of the lesion. A lesion, 2, which involves the nuclei of the 6th and 7th nerves, will produce paralysis of the face and of the external rectus muscle of the eye on the same side, while injury of the as-yet-uncrossed pyramidal fibres to the spinal cord will cause paralysis of the body on the opposite side.

The accompanying diagrams are designed to indicate the site of various local lesions of the brain-stem that give well-defined symptoms. Fig. 55 is a diagram of the level of the decussation of the pyramids. A local lesion occurring in this region is likely to be followed by rapidly fatal symptoms, as important respiratory and vaso-motor mechanisms are almost inevitably disturbed. Lesion 1, situated at the pyramidal

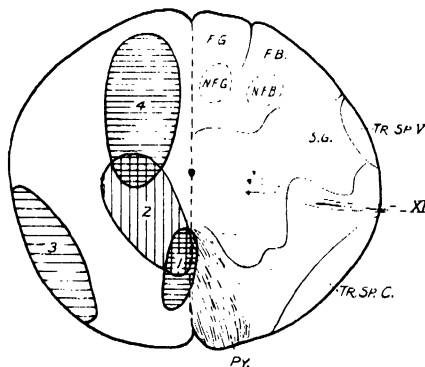


Fig. 55.—Level of pyramidal decussation.

F.G., Column of Goll; F.B., column of Burdach; S.G., substantia gelatinosa; TR SP C., spino-cerebellar tract; TR SP V., vestibulo-spinal tract; PV., pyramidal tract.

decussation, involves necessarily all the pyramidal fibres, and leads to spastic paralysis of all the limbs. Lesion 2, which involves the nucleus of the spinal accessory nerve and the decussation of the pyramids, will cause, in addi

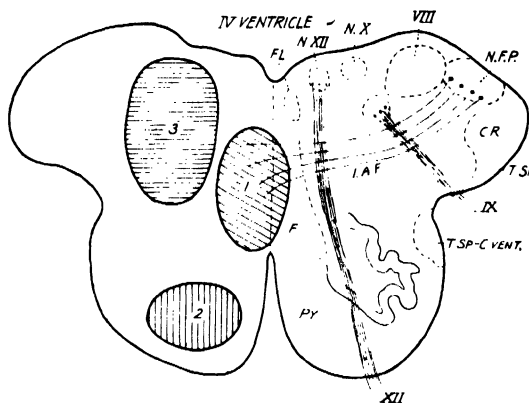
NERVOUS SYSTEM, CENTRAL. LOCAL LESIONS OF

Fig. 56.—Level of decussation of the fillet.

F.L., Posterior longitudinal bundle; N.P.P., nuclei of posterior column; I.A.F., internal arcuate fibres; F, fillet c.w., restiform body; P.V., pyramidal tract; T.SP.-C.DORS., dorsal spino-cerebellar tract; T.SP.-C.VENT., ventral spino-cerebellar tract.

tion to spastic paralysis of the opposite limbs, paralysis and atrophy of the sterno-mastoid and the upper portion of the trapezius on the same side. Lesion 3 involves the spino-cerebellar tracts, and may cause inco-ordination and hypotonia of the muscles of the limbs on the same side. Lesion 4, which destroys the posterior sensory columns and the nuclei of the spinal accessory, may produce dissociated anaesthesia of the opposite half of the body with paralysis of the muscles innervated by the spinal accessory.

Fig. 56 represents the most important local lesions at the level of the decussation of the fillet. Lesion 1 involves the decussating internal arcuate fibres, and thus interrupts the whole of the sensory fibres which convey impulses from the nuclei of the posterior columns to the fillets. The resulting symptoms will be disturbance of those forms of sensibility conveyed by the posterior columns on both sides of the body. Lesion 2, of the as-yet-uncrossed pyramidal tract, just where it is traversed by the fibres of the hypoglossal nerve, produces a spastic paralysis of the contralateral limbs with paralysis and

wasting of the tongue on the same side. It should be noted that whereas in a capsular hemiplegia the tongue is not wasted and when protruded is pushed over to the hemiplegic side, in this case the unilaterally wasted tongue is protruded to the non-hemiplegic side. Lesion 3, besides involving the nucleus of the hypoglossal and thus causing unilateral paralysis of the tongue, destroys the glosso-pharyngeal nucleus and causes loss of taste on the posterior third of the same side of the tongue. A slight extension upwards of this lesion may cause deafness by involving the auditory-nerve terminations in the acoustic tubercle.

Fig. 57 is the diagram of a section at the level of the 7th nucleus. Lesion 1 involves the genu of the facial nerve and the nucleus of the 6th nerve, and produces paralysis of the face and of the external rectus muscle on the same side. Lesion 2, which involves the facial and external rectus nuclei together with the fillet on the same side, gives rise to an atrophic palsy of the

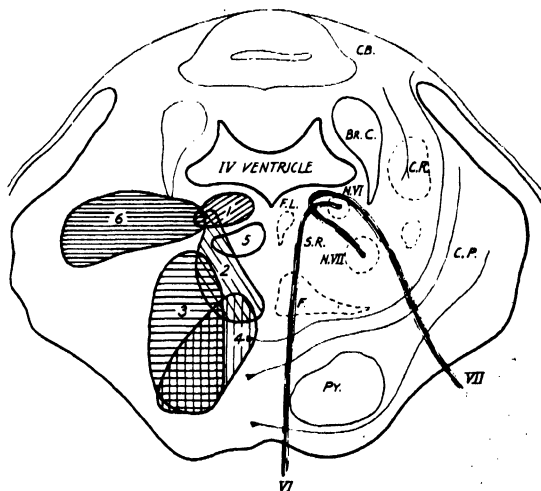


Fig. 57.—Level of facial nucleus.

CB, Cerebellum; BR.C., brachium conjunctivum; C.R., restiform body; S.R., substantia reticularis; C.P., ponto-cerebellar fibres; X., fillet; F.L., posterior longitudinal bundle; PY., pyramidal tract.

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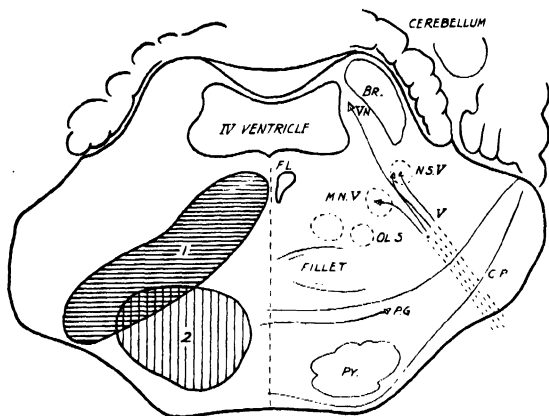


Fig. 58.—Level of exit of trigeminal nerve.

BR., Brachium; V.N., mesencephalic root of nerve; N.S.V., sensory nucleus of 5th nerve; M.N.V., motor nucleus of 5th nerve; C.P., ponto-cerebellar fibres; M.L., superior olive; M.L., posterior longitudinal bundle; PY., pyramidal tract.

facial and external rectus muscles on the side of the lesion, and a dissociated anaesthesia of the body and limbs on the opposite side. Lesion 4 involves the pyramidal tracts as well as the fillet on the same side, and causes a hemiplegia without involvement of the face, together with a dissociated anaesthesia of the body and limbs on the hemiplegic side. Lesion 5, by interfering with the posterior longitudinal bundle conveying the motor fibres of the mesencephalic centre governing the lateral movements of the eyes, will cause a paralysis of the power of conjugate deviation of the eyes to the opposite side. Lesion 6, besides involving the nuclei of the 6th and 7th nerves, extends laterally to interrupt the ponto-cerebellar fibres; if it extends slightly below the plane of this diagrammatic section it will also involve the auditory nucleus. Such a lesion, in addition to causing atrophic palsy of the 6th and 7th nerves, will give rise to cerebellar symptoms and to unilateral deafness.

Fig. 58 is a diagram through the pons at the level of the exit of the trigeminal nerve. Lesion 1 involves the posterior longitudinal bundle, the fillet, and the trigeminal nerve. The symptoms referable to this lesion will be entire loss of sensibility over the trigeminal area on the face, with paralysis of the masseter, temporal and pterygoid muscles on the same side, a dissociated anaesthesia of the body and limbs on the opposite side, possibly some

slight cerebellar ataxy due to the involvement of the ponto-cerebellar fibres, and loss of conjugated lateral deviation of the eyes to the side of the lesion owing to the involvement of the posterior longitudinal bundle. Lesion 2, which lies in the area of the pyramidal tract, will cause a spastic paralysis of the limbs on the opposite side, with paralysis of the trigeminal musculature and anaesthesia of the face on the same side.

Fig. 59 is a diagram of a section at the level of the anterior corpora quadrigemina. Lesion 1, which involves the pyramidal tract in the peduncle, the optic tract, and the lateral geniculate body, will cause a spastic hemiplegia of the opposite side, together with contralateral homonymous hemianopia. Lesion 3 involves the red nucleus and leads to

degeneration of the rubro-spinal tract, and consequently to a wild intention-tremor in the opposite limbs. Lesion 4 is a little more extended, and involves the fibres of the 3rd nerve as they pass through the mesial portion of the red nucleus; therefore, in addition to the contralateral tremor, there will be ptosis, dilatation of the pupil, and paralysis of accommodation, together with paralysis of all the muscles of the eye on the same side, except the external rectus and superior oblique. Lesion 2 involves the fillet and will cause a hemianesthesia of the body and limbs on the opposite side. Lesion 5 involves the pyramidal tract lying in the peduncle, together with the fibres of the 3rd nerve, and produces an ocular paralysis on the side of the lesion and spastic paralysis of the limbs on the opposite side. Lesion 6, which interrupts the pupillo-motor fibres running from the optic tract to the anterior corpora quadrigemina, abolishes the pupillary reaction to a beam of light projected on the homonymous halves of the retinae. Lesion 7 affects the corpora quadrigemina and causes inactivity of the pupils to light, with more or less paralysis of the 3rd nerves if it extends to their nuclei.

Local lesions of the cerebellum.—The cerebellum represents a higher central ganglion of a reflex system that has for function the conservation of the equilibrium of the body both when at rest and in movement. It re-

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ceives stimuli from the muscles, joints, and tendons which register by their interaction the position of the limbs and body, while from the vestibular apparatus it receives impressions which register the position of the head in space when at rest and in movement. The reflex response evoked by these stimuli modifies the innervation of the muscles effected by the cerebrum in such ways as to preserve the general equilibrium of the body both at rest and during movement, and to ensure that the balance of the limb effecting a movement is so preserved as to render that movement effective. The afferent impulses reach the cerebellar cortex for the most part directly, whereas the cerebellar efferent fibres arise from the cerebellar nuclei. Each half of the cerebellum is in connexion by its efferent fibres with the cerebral hemisphere of the opposite side, with both sides of the medulla, and with the homolateral side of the cord.

The afferent impulses from the spinal cord and medulla terminate mainly in the unpaired middle portion of the cerebellar cortex, whilst afferent impulses from the cerebral cortex, from the frontal lobes, the parietal lobes, and above all from the temporal lobes, terminate in the cortex of the contralateral side.

The general symptoms of disease of the cerebellum are described under NERVOUS SYSTEM, PHYSIOLOGY OF. Local lesions of the cerebellum very rarely give rise to symptoms which can be distinguished clearly from those caused by the disturbance of neighbouring structures. Lesions of the vermis are characterized by the typical cerebellar gait and abnormal position of the head, which is often retracted as well as rotated. Movements of the facial and neck muscles are often very sluggish, and speech is slow and staccato. General cerebellar asymnergia is pronounced. Lesions of the lateral cerebellar lobes cause, as a rule, a preponderance of the cerebellar ataxy and dysmetria on the

side of the lesion. Lesions of the cerebellar nuclei are characterized by vertigo, by the "cerebellar attitude" of the body, and by the so-called cerebellar or tonic fits. The accessory cerebellar nuclei are responsible for various forms of nystagmus.

Local lesions of the basal ganglia.—

The optic thalamus represents the terminal ganglion of a system by which painful and thermal stimuli—that is, stimuli which notify the organism of changes that might be injurious to it—are brought into relation with that part of the nervous system which governs the response of the involuntary musculature, the secretory organs, and the vaso-motor mechanism. Lesions of the optic thalamus may give rise to a contralateral hemianæsthesia dolorosa, to loss of control over the emotional reflexes, and in some cases to a mask-like rigidity of the facial muscles, which, however, show no loss of power in voluntary movement. Hemiathetotic movements may also occur on the contralateral side of the body. A crossed hemianæsthesia which affects tactile, pain, and temperature sense much less than deep sensibility has been found occasionally to result from lesions of the thalamus. The loss of deep sensibility leads to ataxy and astereognosis (see Thalamic Syndrome, under PAIN, CENTRAL). Lesions of the lenticular nucleus, when bilateral, may cause bilateral choreiform movements (see LENTICULAR DEGENERATION, PROGRESSIVE).

Lesions of the internal capsule.—The

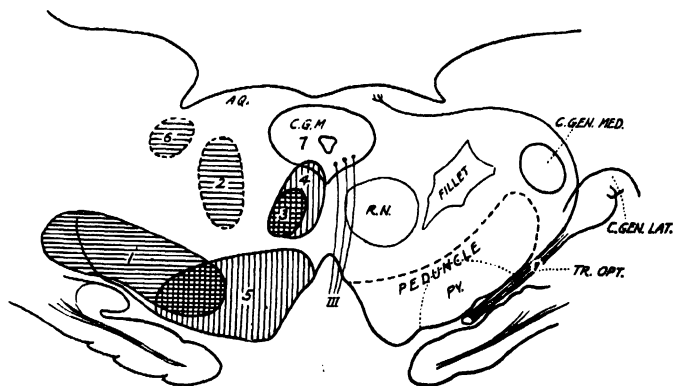


Fig. 59.—Level of anterior corpora quadrigemina.

AQ., Anterior quadrigeminal body; C.G.M., central grey matter; R.N., red nucleus; C.GEN. MED., medial geniculate body; C.GEN. LAT., lateral geniculate body; TR. OPT., optic tract; PY., pyramidal tract.

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internal capsule is the great strand of fibres between the lenticular nucleus externally and the optic thalamus and the caudate nucleus internally. Fig. 60 indicates the order in which the motor fibres from the cortex pass downwards; behind the motor fibres are the sensory fibres, and still farther back are the visual

tions will be involved, causing homonymous hemianopia.

Owing to the manner in which the motor fibres are crowded together in a very small space in the internal capsule, an isolated monoplegia is wellnigh impossible. It should be noted in addition that a lesion of the posterior part of the internal capsule which causes hemianopia and hemiplegia cannot fail to cause a dissociated hemianæsthesia owing to involvement of the intervening sensory tract.

Local lesions of the visual system.—

It is convenient to consider the local lesions of the visual nervous system together, irrespective of their relation to different parts of the brain. Plate 22 is a diagram of the relations of the visual neurones. Lesion A, by interrupting all the fibres in an optic nerve, causes homonymous blindness. Lesion B, at the optic chiasma, causes bilateral temporal hemianopia; while lesion C, at the outer side of the chiasma, by which the uncrossed fibres alone are affected, causes a loss of vision in the nasal field of the homonymous eye. Such lesions are occasionally bilateral; they may be due to a retro-chiasmal tumour pushing the tracts forwards against the unyielding carotid artery. Lesion E, which represents a complete interruption of an optic tract, produces an homonymous hemianopia, the temporal half of the retina on the side of the lesion and the nasal half of the opposite retina having their visual neurones interrupted; and as the afferent neurones from these portions of the retina to the pupillo-motor centres are also involved, a ray of light on the blind halves of the retina will not cause contraction of the pupils (*Wernicke reaction*). Lesion F, which affects the primary optic centre in the lateral geniculate body, also produces an homonymous hemianopia. Lesion H involves the primary centre for the pupillary afferent fibres in the anterior corpora quadrigemina and causes loss of the pupillary reaction to light but not to other stimuli, without impairment of vision. Lesion G, in the optic radiation, causes homonymous hemianopia without impairment of the pupillary reactions. A lesion of the visual cortex, 8, likewise causes an homonymous hemianopia if the entire area is involved. Inasmuch as the cortex above the calcarine fissure receives impulses from the upper portion of the retina, while the visual cortex below the calcarine fissure is supplied by the lower portion of the retina, it follows that a local lesion of the supracalcarine portion causes a blindness of the lower quadrants

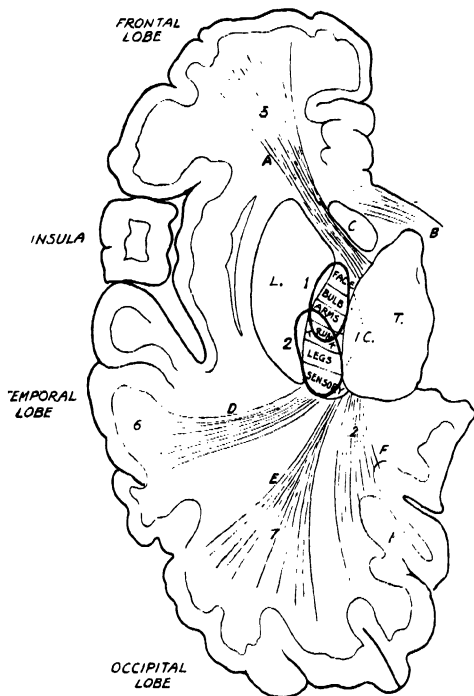
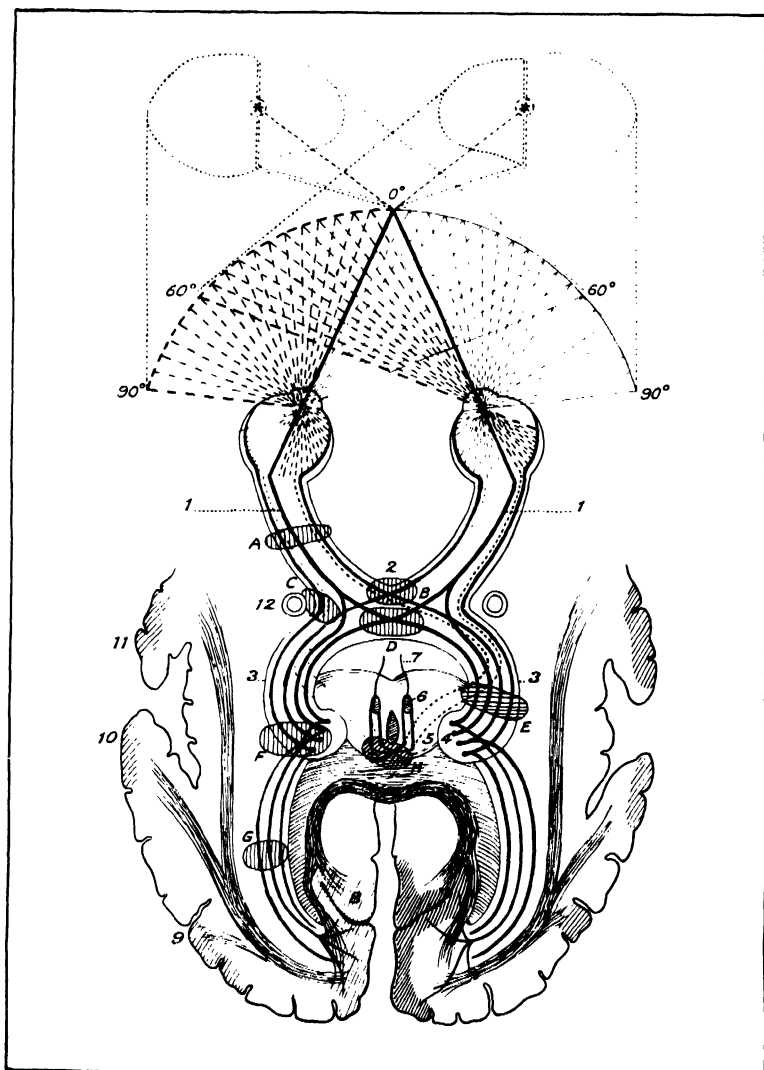


Fig. 60.—Horizontal section through the internal capsule.

I.C., Internal capsule; T., optic thalamus; L., lenticular nucleus; C., caudate nucleus; A, fibres from frontal lobe to pons and thalamus; B, corpus callosum; C, acoustic fibres to temporal convolution; D, occipito-temporal pontine, and thalamic fibres; E, visual fibres to occipital lobe

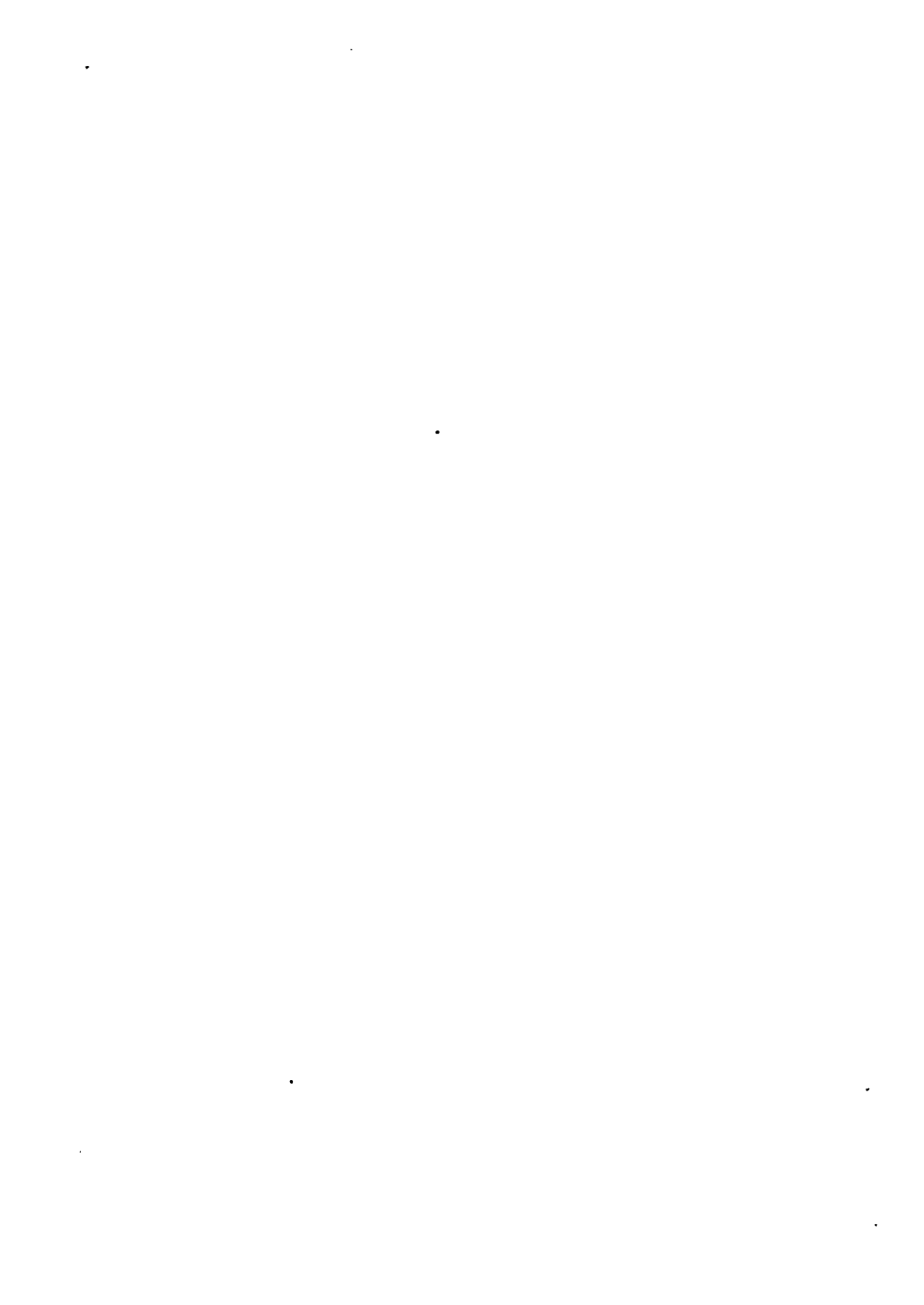
fibres, on their way to the visual cortex. Lesion 1 in Fig. 60 causes paralysis of the face and of the arm, and possibly less complete paralysis of the leg, on the contralateral side of the body. Lesion 2 affects the arm a good deal less than the leg, and, owing to involvement of the sensory fibres, it also causes a crossed hemianæsthesia. If the lesion extends farther back the fibres of the optic radia-



1, Macular fibres. 2, Chiasma. 3, Optic tracts. 4, Lateral geniculate body. 5, Anterior corpora quadrigemina. 6, Oculo-motor nucleus. 7, Oculo-motor nerve. 8, Visual cortex. 9, Visual speech centre. 10, Auditory speech centre. 11, Motor speech centre. 12, Carotid artery.

PLATE 22.—LESIONS OF THE VISUAL SYSTEM (see Text).

(Based on diagram in de Lapersonne and Cantonnet's "Manuel de Neurologie Oculaire.")



NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF

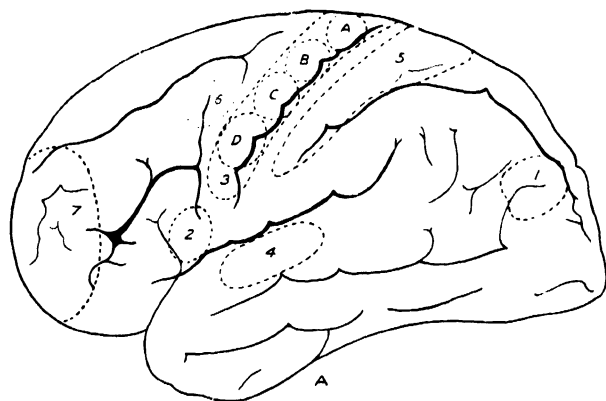


Fig. 61.—Diagram of left side of cerebrum, outer surface.

of the homonymous visual fields, whilst a lesion of the subcalcarine visual cortex causes an upper quadrantic hemianopia. In none of the forms of hemianopia so far discussed has the macular or central area of the field of vision been involved; this may be accounted for by the assumption of a bilateral distribution of the macular fibres to each optic tract and cortex. A bilateral affection of the entire visual cortex leads to complete blindness, but the pupils react normally to light. A lesion of the optic association tracts running to 9—the angular gyrus—may cause disturbance of the power of reading, word-blindness, and occasionally conjugate deviation of the eyes to the side of the lesion. Interruption of all the association fibres from the visual cortex will cause, if the lesion is bilateral, psychical blindness with conservation of vision.

Focal lesions of the cerebral cortex.—A lesion of the cerebral cortex may give rise both to irritative symptoms and to symptoms of loss of function. Figs. 61 and 62 are diagrams of the outer and inner surfaces of the left hemisphere. The former will first be considered. Lesion 1 is situ-

ated in the angular gyrus; an irritative focus here causes deviation of the eyes to the opposite side, a lesion entailing loss of function causes deviation to the side of the lesion. If the lesion is on the left side in a right-handed person it may cause word-blindness and alexia. Lesion 2, in the 3rd frontal convolution, if on the left side in a right-handed person, may cause motor aphasia. Lesion 3 extends over the whole of the Rolandic area or motor cortex. An irritation here gives rise to convulsions beginning in and sometimes

limited to the right side, whilst a destructive lesion produces hemiplegia of the right side. A subcortical lesion underlying this and interrupting the cortical motor neurones would cause similar symptoms. The area subtended by Lesion 3 has been divided into a number of subsidiary areas. Lesion A would affect the leg, B the upper extremity, C the hand, D the face and tongue muscles. Further subdivision of the motor areas would be justified on experimental rather than on clinical evidence. In each case the irritative effect of the local lesion would be to cause a Jacksonian fit beginning in the muscle-group concerned, and the deficiency effect would be weakness of the

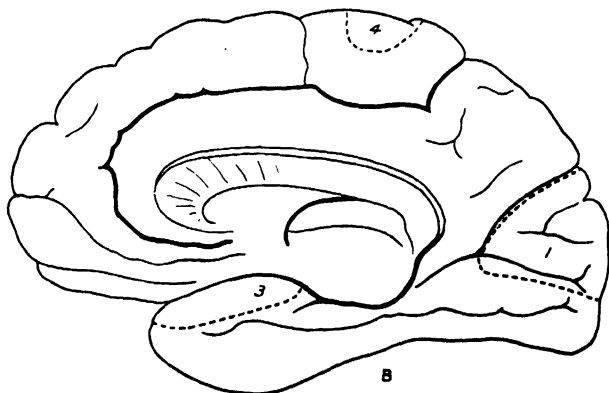


Fig. 62.—Diagram of right side of cerebrum, inner surface.

corresponding muscles. Lesion 4, in the upper temporal convolution, if situated on the left side, causes sensory aphasia. Lesion 5, in the parietal post-Rolandic area, causes sensory disturbances which are situated on the contralateral side of the body. Local lesions of smaller extent included in this area would cause contralateral anaesthesia of a cortical type of the extremity to the motor area of which they were in juxtaposition. Irritative lesions of the sensory area may give rise to convulsions, beginning with sensory subjective disturbances in the contralateral peripheral area which they subtend. Local lesions of the sensory cortex never lead to complete loss of sensation.

Lesion 6, at the foot of the second frontal convolution, involves that portion of the cortex which has control over the lateral movements of the head and eyes to the opposite side. An irritative lesion will excite conjugate deviation of the head and eyes to the contralateral side, whilst a lesion causing abolition of functional activity will cause the conjugate deviation towards the side of the lesion. Lesion 7 is in the frontal lobe. Most of its known symptoms are referable to the involvement of adjoining cortical areas, but the following symptoms have been repeatedly noted in frontal tumours though they are very inconstant, viz. loss of memory, general confusional states, a curious form of childishness characterized by silly attempts to be smart at the expense of others, an occasional fine tremor in the hands, blindness owing to direct pressure of a frontal tumour on the optic nerve, and unilateral anosmia due to similar pressure on the olfactory bulb.

On the inner aspect of the brain (Fig. 62), Lesion 4 in the paracentral lobule involves the motor centre for the toes and ankles; irritation of it will be manifested by Jacksonian epilepsy beginning in these parts, while loss of function causes weakness of all their movements. A destructive lesion at 1, by damaging the visual cortex, produces homonymous hemianopia, whilst an irritative lesion may give rise to the sensation of coloured lights flashing in the homonymous hemianopic field, which may be the aura of a generalized fit. Lesion 3, in the hippocampal region, may cause, as an irritative phenomenon, sensations of taste and smell, with the "dreamy states" which are described in the article on EPILEPSY. Loss of taste and smell from such a lesion is a very rare occurrence.

F. L. GOLLA.

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—The nervous system is composed of a number of nerve units which are anatomically and genetically independent of one another. These units are called *neurones*. The neurone consists of the nerve-cell from which are given off a number of protoplasmic processes. One of these processes (more in certain types of neurone) becomes the *axis-cylinders* of the nerve-fibres, which proceed to their final destination, and there break up into a plexus of fine branches. During their course they may give off several collateral fibrils, which also end in terminal arborizations. The other protoplasmic processes form very complex arborizations soon after leaving the nerve-cell, and are known as *dendrites*. These arborizations generally terminate in a mesh formed with the processes of other neurones. Each neurone is independent, and has no anatomical continuity with any other cell structure.

Physiologically the nervous system constitutes a mechanism by which chemical and physical changes taking place in any given cell or collection of cells are communicated to other spatially separate cells. It is not the sole means of communicating chemical and physical changes, since products of activity are set free by every cell into the blood and lymph, and these chemical bodies may influence other cells by altering their chemical or physical equilibrium. Whilst, however, the distribution of chemical excitants by the blood involves the stimulation of all such cells as are susceptible of being acted upon by the excitant in question, by means of the nervous system a change occurring in a single cell or group of cells may be communicated to another cell or cell-group to the exclusion of all similar cells that are not teleologically essential to the specific response of the organism to the stimulus. The recognition of the communication of changes of physical state from one system to another as the sole function of the nervous system considered physiologically, does not commit us to any theory as to its relation to psychical processes. Physiology is only concerned as an objective science with the description and measurement of objective changes. That the integrity of certain portions of the nervous system is necessary to the existence of consciousness, as we know it, appears to be an undoubted fact, but as physiologists we have no right to express an opinion as to the existence of any causal relation between consciousness and nervous activity, or as to the possibility

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of consciousness existing independently of neural activity. The nervous system is, then, physiologically considered, a mechanism for the communication of changes of physical state between cells not otherwise in direct communication with one another.

Specific functions of the neurones.

—Axis-cylinders communicating with non-nervous cellular elements constitute the peripheral nervous system, while neurones which act as the means of communication between other nerve-cells form the central nervous system. The physical or chemical disturbance communicated by the neurone is spoken of as a nervous impulse. The peripheral nerves are composed of bundles of axis-cylinders which may either be centripetal fibres conducting impulses from the periphery to the central nervous system, or centrifugal fibres conducting impulses from the central nervous system to the periphery.

The *centrifugal nerve-fibres* may conduct impulses exciting movement to contractile tissue, impulses exciting secretion to the secretory cells, and inhibiting impulses which arrest cellular activity. The *centrifugal nerve-fibres* acting on striated muscular tissue end in terminal arborizations in close contiguity with the muscle-fibres; they arise from nerve-cells lying in the central nervous system. The secretory fibres and those acting on non-striated muscle and the corresponding inhibiting fibres arise from nerve-cells which lie outside the central nervous system. This system governing the automatic structures is spoken of as the vegetative nervous system (q.v.).

The *centripetal nerves* conduct from the periphery impulses that may give rise to conscious sensation, and others which do not cause changes in consciousness. A sharp distinction cannot be made between these two types of centripetal nerves, since the production of conscious sensation is in some cases dependent on the intensity of the stimulus.

The *nerve-cell body* is the nutritive centre of the neurone. On severance from it the axis-fibre perishes (Wallerian degeneration). From the cell-body fresh axones may in certain circumstances be developed when the axone has been destroyed. Apart from its function of preserving the nutritive equilibrium of the neurone, there is no reason to think that the cell-body takes any specific part in the transmission of impulses. It has been found possible, on account of its size, to remove the cell-body in a ganglion of the crab without

conduction through the neurone being affected.

Physiological stimulus of the neurone.

—The transmission of a nerve impulse is generated in a neurone by external stimuli affecting the receptor cell about which its terminal arborization is formed. The receptor cell is adapted to receive certain specific forms of stimulus; thus, the retinal end-organs are adapted for light radiations, the cells of the organ of Corti for sound vibrations, and so on. No matter how such a receptor cell is stimulated, the impulse generated in the neurone causes the sensory impression that is specific for that type of cell. Thus, electrical stimulation of a retinal receptor will always be interpreted as light, that is, in terms of the normal physiological stimulus. Further, direct stimulation of the neurone will always excite the same type of sensory impression as that which is specific to its receptor cell; thus, electrical or mechanical stimulation of the optic nerve will arouse the sensation of light. This does not necessarily mean, as is sometimes assumed, that the type of impulse passing along the neurone of a specific sensory nerve differs from that passing along another arising in a different type of receptor; it is probable that the impulse is identical in nature, but that its interpretation as light or as sound is a function of the particular neurone or group of neurones in the cortical grey matter to which it is transmitted. Whilst the receptor cell is susceptible to minimal stimuli of a nature to which the organ that it belongs to is adapted, it is only with difficulty excited by stimuli of a different nature.

A nerve impulse may be generated in a neurone by changes taking place in other nerve-cells, the terminal arborizations of which are contiguous to those of the neurone excited. This occurs when a nerve impulse is communicated from one neurone to another by the mediation of a third intercalary neurone. A neurone may be directly stimulated by changes in the blood or tissue fluids in direct contact with it; stimulation by this method is not necessarily due only to changes in the vicinity of the terminal arborizations, but may occur at any point of the course of the neurone.

Nature of the nerve impulse.—The velocity with which a nervous impulse travels along a mammalian motor nerve is about 120 metres per second. Such a speed of propagation is strong evidence of the physical

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nature of the nerve impulse. A refractory period, lasting about one-thousandth of a second, follows the passage of a nerve impulse, and during this period the nerve does not respond to another stimulus. Compared with muscle the axone may be said to be indefatigable, since if a motor nerve be stimulated for hours whilst the impulse is blocked from arriving at the muscle either by curare or electrotonus, when the block is removed the nerve conducts excitatory impulses without any appreciable alteration in efficiency. The most delicate

been shown to be true of the motor axone, and is probably equally true of the afferent fibre. The nature of the intercommunication between the neurone and other cells is not understood. There is no anatomical continuity either between the neurones themselves, or between the neurone and a receptor or motor cell, in vertebrates at least, though in some of the invertebrates there is evidence of anatomical continuity between the neurones. Whether there be an actual spatial discontinuity, as microscopic preparations seem to show, or

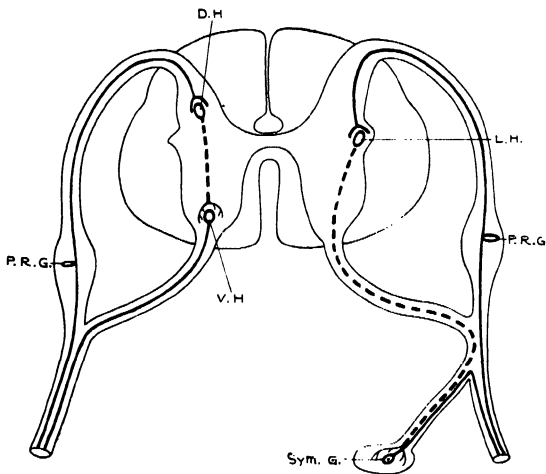


Fig. 63.—Reflex paths. On the left, an ordinary spinal reflex; on the right, a reflex of the vegetative nervous system. (After Gaskell, "Involuntary Nervous System." By permission of Messrs. Longmans, Green & Co.)

D.H., Posterior horn cell; L.H., lateral horn cell; V.H., anterior horn cell; P.R.G., posterior root ganglion; SYM.G., sympathetic ganglion.

measurements fail to demonstrate any evolution of heat when an impulse passes down a nerve-trunk. The passage of a nerve impulse down a nerve is accompanied by an alteration of the electrical state of the nerve, the portion of nerve affected by the excitatory state becoming electro-negative to the portion unaffected. This propagation of a change of electrical state occurs *pari passu* with the passage of a nerve impulse; it is, however, an open question whether the current of action, as it is called, is the sum of the physical changes constituting the nerve impulse or a concomitant accessory phenomenon. The nerve impulse is conducted in both directions; this has

whether the terminal arborizations of the neurones are in actual physical contact with each other, cannot be determined with certainty. The nexus between neurone and neurone, whatever its objective character, is conveniently designated by the term *synapse*. Similarly, it is convenient to speak of the nexus between the nerve-ending and the muscle-fibre as the *myoneural junction*, without committing oneself to any hypothesis as to its objective nature. These terms have a definite physiological significance, in that at the synapse certain modifications of the nerve impulse appear to take place. The part probably played by the synapse in the intercommunication of nerve impulses will be clearer when the reflex is studied.

The reflex.—A reflex is, in its simplest terms, the conduction by means of a neurone of a stimulus received by a receptor cell from the environment to another cell responsive to the stimulus. In the vertebrate nervous system a simple reflex always involves the mediation of three neurones—an *afferent* neurone conveying the impulse from the receptor cell, an *intermediate* neurone communicating the nerve impulse to the *efferent* neurone by which it is conducted to the responsive cell. In a simple reflex involving the communication of a stimulus received from the environment to a skeletal muscle, the intercalary neurone and the cell-body of the efferent neurone lie within the cerebro-spinal axis. In a reflex of the vegetative nervous system in which the responsive cell is a non-striated muscle or a secretory cell, the cell-body of the efferent

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neurone lies outside the cerebro-spinal axis, and the greater portion of the axone of the intercalary neurone running to the efferent neurone also lies without the central nervous system. Fig. 63 illustrates both the anatomical difference and the anatomical similarity between the reflex arcs of the voluntary and the vegetative nervous systems.

A simple reflex is, however, an abstraction. Just as there can be no such thing as a pure perception, inasmuch as we never perceive anything without remembering, so no nerve impulse can arrive from the environment and be communicated to a responsive cell by the mediation of an intercalary neurone without affecting the distribution of energy in other neurones of the nervous system which are in physiological communication with the mediate and afferent neurones. However great the complexity of the inter-relations of a reflex arc with other neurones in the central nervous system, the impulse arising from any one receptor cell and stimulating any one responsive cell can only enter the nervous system by a specific afferent neurone and leave it for the responsive cell by a specific efferent neurone. It is convenient, therefore, in dealing with the general physiology of the nervous system, to consider first the mechanism of a simple reflex abstracted from its relations to other reflex arcs.

Our knowledge of reflex physiology and of the linking-up and co-ordination of reflexes owes a very great deal to the work of Sherrington; in dealing with reflex nervous physiology much of what follows is largely indebted to his exposition. If conduction in a nerve-trunk be compared with that in a reflex arc, certain differences will be noted. (1) A longer period elapses between the application of a stimulus and the response when the afferent portion of a reflex arc is stimulated than when a nerve of the same length is stimulated. This delay does not take place in the nerve-cell, since measurements of the velocity of a nerve impulse when the posterior roots are stimulated centrally and peripherally to the spinal ganglion show that there is no loss of time in the passage of the impulse through the ganglion cell, and the delay is therefore probably referable to the synapses between the afferent and the efferent neurones and the intermediate neurone. It might appear that the increase of time was utilized in establishing a path of communication in the synapse, but experiments show that if, when a reflex is

already in being and therefore the path is open, a sudden increase of the stimulus be effected, the latent period before a corresponding increase of the end-effect is observed is the same as that between the initial stimulus and the initial response. (2) When a motor nerve is stimulated the response of the muscle ceases within a very short period of the cessation of the stimulus, and this is not affected by the strength of the stimulus. When a muscle is stimulated through a reflex arc the response is prolonged for an appreciable period after the cessation of stimulus, and the stronger the stimulus the longer the period of after-discharge. This effect has been aptly compared to the persistence of a positive after-image left by a visual stimulus. It is a matter for speculation whether this effect is caused by inertia at the synapse, or a repercussion of the effect among other neurones communicating with the intermediary neurone. (3) Summation of subliminal stimuli so that they become effective does not occur when a muscle is stimulated through its motor nerve, though there is some evidence of a process of summation in the case of secretory nerves. Summation is a characteristic of reflex conduction. Thus, in the scratch reflex of the dog evoked by stimulation of a certain area of skin on the back, a single stimulus below the threshold intensity was found, which only on its fortieth repetition, and nearly four seconds after its application, became effective and provoked the reflex (Sherrington). (4) Conduction of a nerve impulse in a nerve-trunk has been shown to take place in both directions; in a reflex arc, conduction takes place in one direction only; thus, stimulation of the efferent motor neurone of the arc never gives rise to a sensation by a reversal of the normal direction of the reflex flow. This characteristic is referable to the synapse, inasmuch as in such lower organisms as *Medusa*, where there appears to be anatomical continuity between the neurones, conduction in a reflex arc may take place in both directions. (5) The number of nerve impulses produced in a nerve by repeated stimuli corresponds closely with the number and rhythm of the stimuli so long as the frequency of the stimuli is not so great that they are repeated within the refractory period of the nerve, a period of about one-thousandth of a second. The discharge of a reflex arc is rhythmical even under a constant stimulus, and the existence of rhythm is due to the occurrence of a refractory phase during

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which the stimulus is incapable of exciting a fresh nerve impulse. In the dog, after transection of the spinal cord at the neck, the scratch reflex of the hind leg can be evoked with certainty by stimulation of a saddle-shaped area of skin on the back. This reflex is a rhythmic alternate flexion and extension at hip, knee, and ankle. Each flexion recurs with a frequency of about four times a second. It has been found by Sherrington that under any form of excitation (heat beam, constant current, induced current, high-frequency current, and mechanical stimuli) the rhythm of the flexor response remains almost the same, nor can the refractory phase occurring between the beats of the flexor reflex be overcome by increasing the intensity of the stimuli. This power of the reflex arc of responding by a rhythm of its own without reference to the frequency of the stimulus is inherent to a large number of reflexes which are connected with the gait or the removal of noxious stimuli; that is, when continual, repetition of the movement by alternation of an active and a refractory phase, during which the muscles are relaxed, is of advantage to the organism. (6) While the magnitude of the response evoked by stimulation of a nerve-trunk varies within certain limits with the strength of the stimulus, the correspondence between intensity of external stimulus and the reflex arc effect is very limited. Many reflexes respond only with a maximal effect to any adequate stimulus, but others show a limited range of correspondence between magnitude of response and stimulus.

The differences that have been so far considered between the reaction of the nerve and the reflex arc put us in possession of some of the functional characteristics of the central nervous system. Though essentially constructed of the same conducting material that has been studied in the axone fibre, the reflex arc transforms the impulse arriving by the afferent fibre in such a manner that it will evoke an end-effect appropriately adjusted to the object to be attained by the reflex. It has been already said, however, that an isolated simple reflex does not occur in the mammalian body. The afferent impulse, once it has been communicated to the intermediate neurone, is not simply conducted in an altered form to the efferent neurone. There may be a single intermediate neurone or there may be many, but in any case the mediate system is in relation by means of its synapses with other reflex arcs. There is no reason to believe that a selection of the path

of transmission of a nerve impulse takes place within the neurone unit; if there be no such selection a stimulus communicated to the dendrite of a neurone will be conducted to one and all of the terminal arborizations of the axone, and hence be susceptible of communication to all the neurones with which it may form synapses. These other neurones are concerned either directly or mediately with other reflex arcs, and hence the stimulation of a reflex necessarily influences other reflexes whose neurones are either directly or indirectly in physiological communication with it. It will be at once apparent that, if there were no directive mechanism to effect a limitation of the spread of the excitation, the stimulation of an afferent fibre might cause a reaction of the entire nervous system, since each neurone is in physiological contact with every other neurone by a more or less indirect path. One condition that limits this spread has been already alluded to, that is, the irreversibility of conduction in the reflex arc. We have seen that there is every reason to think that the directive mechanism which permits or rejects the acceptance of a nerve impulse lies in the synapse. The synapse might be compared to a valved opening in a system of tubes that allows the passage of fluid in one direction only. There is, however, another factor which ensures the relative limitation of the spread of the reflex response to such proportions as will adequately effect the bodily adjustment to the stimulus. We have seen, when considering the simple reflex, that a stimulus that is inadequate to provoke a response may, on being repeated several times, be effective. That is, the synapse is able to offer a resistance to a stimulus which, applied to the efferent neurone, would in itself be effective, but this resistance gives way before a repetition of the stimulus. Hence, if a stimulus be answered by an adequate reflex response, nothing further may happen should it be insufficiently strong to overcome the resistance of the collateral synapses, but if the stimulus continues or is of greater magnitude it may lead to the involvement of other reflex arcs.

Inhibition.—The displacement and the replacement of any part of the body by muscular contraction involves the successive contraction of two muscles, or groups of muscles, acting in mutually opposing directions. Sometimes the replacement will be effected to a great extent by gravity, but this will only

act when the displaced part is in a particular relation to the lines of the earth's attraction. If the two opposing muscular groups are attached to the structure involved, it is necessary, if any movement is to take place, that only one muscle-group should actively contract, and that if the opposing group be in a state of active contraction its activity should be inhibited. One effect, therefore, of the calling into activity of a reflex arc will be the inhibition of the activity of other reflex arcs that would tend to interfere with its end-reaction. The nerve impulse communicated to the synapses of the neurones belonging to such opponent reflex arcs will therefore have the effect of inhibiting any excitatory process that may be present. Inhibition takes place in the neurone itself. In vertebrates there is no evidence of the existence of inhibitory nerves directly influencing the activity of non-nervous structures. The mechanism of inhibition is not understood. It is not simply the removal of stimulus, such as occurs when we switch off the current from electrodes stimulating a peripheral nerve, for we find that after inhibition an effective stimulus gives rise to an increased response. Further, it has been shown that cessation of a stimulus applied to a reflex arc is followed by an after-discharge lasting an appreciable time, whereas the cessation of the end-effect depending upon inhibition is practically instantaneous. It would appear that whereas the excitation of a neurone leads to a discharge of its potential energy, i.e. a process of dissimulation, the inhibition of an active neurone leads to an increase of potential energy, which is a process of assimilation, as witnessed by the rebound effect on cessation of the inhibiting process.

Interference.—When the discharge from two reflex arcs has a directly antagonistic effect, the preponderant stimulus acting on the one inhibits the activity of the other arc. It may happen that two reflexes both make use of the same efferent neurone. Thus, in the case of a dog whose spinal cord has been transected, two reflexes can be elicited, both of which employ the same group of muscles—the flexors of the knee, namely the *scratch* reflex, elicited by stimulation of the skin of the back, causing a rhythmic contraction of the knee flexors; and the *flexion* reflex, in which a tonic flexion of the knee is maintained in response to stimulation of the foot. When both these areas are stimulated simultaneously the scratch reflex is abolished and the

flexion reflex prevails. If now the stimulation of the foot be discontinued, the scratch reflex reappears. Under certain conditions this interaction may be reversed and the scratch reflex be caused to prevail. It is this phenomenon that has been termed *interference*; there is no inhibition of the efferent neurones to the flexors of the knee, but the application of a stimulus through one set of neurones has made these inexcitable to a stimulus arriving from another set. It is not possible as yet to indicate the mechanism by which this interference is effected. Assuming it to take place at the synapse, we can merely say that excitation of a neurone through one synapse may render it less excitable to excitation through another synapse, and vice versa, so that the victory in the struggle will be to the prepotent stimulus. There are, however, certain difficulties in this interpretation, as the end-effect of the preponderant reflex stimulus does not exhibit the diminution of strength that we should expect if interference were due to a mutual increase of resistance at the respective synapses.

Tonus.—In considering the interaction of reflexes there is another factor besides the active contraction of groups of muscles. A skeletal muscle is normally maintained in a condition of slight tonic contraction. There is some reason to think that this tonic contraction depends on excitation of the sarcoplasm as distinguished from the anisotropic portion of the muscular substance. The maintenance of the tonic contraction depends on the activity of the efferent motor neurone, for if the motor nerve be severed the muscle relaxes completely. The tonus-maintaining activity of the motor neurone, again, depends on its stimulation by impulses constantly conducted from the periphery by the afferent neurones of its reflex arc, for when these are interrupted by cutting the posterior roots the tonus disappears. Tonus, then, is a constant reflex discharge affecting the muscle in such a fashion as to cause a slight degree of contraction, and its degree can be increased or diminished by variations in the intensity of the peripheral stimulus. It is obvious that just as an active contraction in an antagonistic muscle must be inhibited in order that a movement may be effected, so the tonus of an antagonist will have to be modified. Certain experiments have shown that an inhibition of tonus in the antagonistic muscles takes place when an active contraction of their opponents is elicited, but this is by no means invariably the case. An

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actual increase of tonus of the antagonists may occur, thus permitting the slow and smooth performance of what would otherwise be a jerky movement. Apart also from the actual antagonistic muscles, others are involved in a reflex movement, their function being to prevent displacement of the parts moved in relation to the body, except in the direction immediately conditioned by the actively contracting muscles. Thus, when the thumb is abducted the flexor carpi ulnaris may be felt to contract slightly in order to prevent the hand being pulled over to the radial side. Such muscles are termed *synergic muscles*; they may act either by a definite active contraction as in the case instanced, or by an increase in their degree of tonic contraction. A reflex discharge, therefore, may not only excite or inhibit active contraction in other reflex arcs, but may similarly cause increase or decrease of tonus.

In the reflexes of the *vegetative* nervous system the non-striated muscle is only capable of tonic contraction, hence this distinction does not appear. The efferent neurone which corresponds to the intermediate neurones of the spinal cord is in relation to two reflex systems, the sympathetic and the parasympathetic. These systems have a mutually antagonistic action, and each is capable of producing inhibition of end-organs; but, unlike the central nervous system, where the final common path to the effector cell is a single efferent neurone, we find separate efferent neurones for each system, and the mutual inhibition appears to be effected at the terminal nexus between the efferent fibres and the effector cell, be it non-striated muscle or gland cell.

Interconnexion of the reflexes.—The co-ordinating mechanism of a single reflex has already been considered, together with the manner in which the excitation of a reflex influences other reflex arcs. It now remains to consider the mechanism by which appropriate reflexes are co-ordinated to perform complicated movements. The prevalence of one type of reflex over another when both types make use of the same effector mechanism is due to the nature and intensity of the stimulus. A stimulus that arises from a disturbance causing injury to the tissues will always tend to gain possession of the field in competition with an innocuous stimulus. Given two or more stimuli of like nature affecting different receptor areas, the effector mechanism will be

utilized by the reflex arc submitted to the stronger stimulus. The anatomical interconnexion of the reflex arcs is such that, when a reflex excitation takes place, those reflex arcs which tend to intensify or propagate movement directed to the same end as that towards which the original reflex tends, will be rendered more easily excitable, they will be therefore more readily called into activity by stimulation of their specific receptors. Again, when the reflexes are of antagonistic nature, the inhibited reflex will be found, on cessation of the stimulus causing the activity of its antagonist, to be in a condition of hyperexcitability, so that it will be readily evoked by a slight stimulus. The advantage of this mechanism, which has been termed *successive induction*, is obvious in an ordered sequence of antagonistic movements such as occur in walking. In this connexion a point that is often overlooked may be alluded to—the fact that the receptors lying in the effector organs themselves are those which are most potent in the excitation of the successive reflex. Thus, the sensory nerves from a contracted muscle will tend to excite the reflex contraction of its antagonists. The whole subject is, of course, difficult to investigate experimentally, but the prediction may be hazarded that the part played by the receptors of excited end-organs in the ordering of reflexes will be found to explain much that is at present referred to the interaction of connecting neurones.

Control of reflex action by the cerebral mechanism.—Up to this point we have discussed in general terms the co-ordination and inter-relation of the simple reflexes of the spinal cord. Considered physiologically, the central nervous system is but a compound of reflex arcs, some of them relatively simple, others exceedingly complex. From the physiological standpoint the nervous system has no other function than that which has been defined as constituting reflex action. But whereas in the reflexes which have been considered the end-effect is apparent and follows more or less swiftly on the stimulus, whether it manifests itself as a specific response or as the modification of the response of other reflex arcs, in the more highly complicated reflexes of the cerebrum the end-effect may be delayed almost indefinitely so that it is no longer apparent. A word may be spoken and the resulting stimulus conducted to the auditory centres, but though the subject give no perceptible reflex response the same type of mechanism

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as that already studied in the spinal reflexes has been set in motion. The auditory stimulus may have lowered or raised the excitability of the neurones of other reflex arcs, or it may have simply succeeded in modifying the excitability of the neurones concerned in its own path, without, however, penetrating to the efferent motor neurones. In any case, it has caused some modification of the reflex mechanism. Has the nervous system no other function than to act as a link between certain receptor and effector cells? It must once again be emphasized that as physiologists we are attempting to investigate physical changes in terms of physical measurements; the moment we cease to do so we cease to talk the language of physiology. Though we may note, and even find it convenient to note, conscious perceptions that accompany physiological changes, we must never lose sight of the fact that we are then making use of a method that is not physiological. Since, however, we know less of the reflexes evoked by most afferent stimuli than of the sensations we experience, we are often forced to speak of them in terms of sensation.

The afferent system.—Taking the nervous system as a compound of reflexes, it is now necessary to study in general terms the physiological arrangement of afferent impulses. From the skin arrive impulses which, according to their sensory characters, are grouped as tactile, pain, and thermal sensations. Beneath the skin, independent of all touch and pain spots, lies an afferent system whose primary function appears to be the communication of disturbances of tension in the tissues in which its receptor cells lie. Pressure such as that evoked by a touch can be appreciated and localized; increase of pressure causes pain. From receptors in the muscles, joints, and tendons impulses are conducted which excite postural reflexes, and by means of which we gain our knowledge of the posture of the limbs. If we translate the psychical equivalents of these different impulses into terms of their relation to the end-effects, then this group consists of stimuli which do not menace the integrity of the tissues but serve as signals for bodily readjustment. These two groups of afferent impulses, after entering the cord by the posterior spinal roots, pass upwards by distinct anatomical systems. The painful and thermal impulses ascend the spinal cord by a crossed secondary path lying in the ventrolateral column, the tactile and postural im-

pulses by an uncrossed path in the dorsal column. Thus the afferent impulses entering the spinal cord may be divided into two systems, from the anatomical, the psychological and the physiological point of view. The afferent impulses from the viscera likewise enter the spinal cord by the posterior roots, and are probably associated in their course with the pain and thermal impulses. The fibres conducting postural and spatial impulses run through a longer intraspinal course than those conducting pain impulses, and give off afferent impulses to the spinal and cerebellar co-ordinating mechanisms that lie in the same side of the cord. In the rest of its headward path the afferent impulse runs through at least one secondary neurone. The system conducting the postural and spatial impulses ultimately decussates in the lower part of the medulla, and the two afferent paths, both of which are now crossed, terminate in the optic thalamus. Here a regrouping takes place. Those impulses connected with pain and thermal sensations are primarily concerned with the preparation of the body as a whole to encounter stimuli that may be injurious. This preparation or defensive mobilization of the body as a whole is co-ordinated in the thalamus, and will be presently considered under the term of emotional reflexes. The thalamic centre of generalized organic reflexes is in connexion with the cerebral cortex, which appears to have some power of controlling its activity. The loss of the possibility of evaluating degrees of heat and cold, or of accurately localizing painful sensations when the cortex is injured, shows that the degree of excitation and response of the thalamus influences the cerebral cortex. The postural and spatial impulses have also thalamic connexions, as might be expected inasmuch as no sensation can be completely free from emotional tone, but their chief function is to find in the cortex those connexions that will allow them to evolve or modify complicated and discriminatory reflexes of the skeletal musculature by co-ordination with afferent impressions arriving from the organs of special sense, e.g. vision, hearing, and smell. The psychical equivalents of these functions bear out this view of their distribution, for we find that a destructive lesion of the cerebral cortex lying behind the fissure of Rolando is accompanied by disturbance of tactile sensibility, of discrimination of points touched and degrees of temperature,

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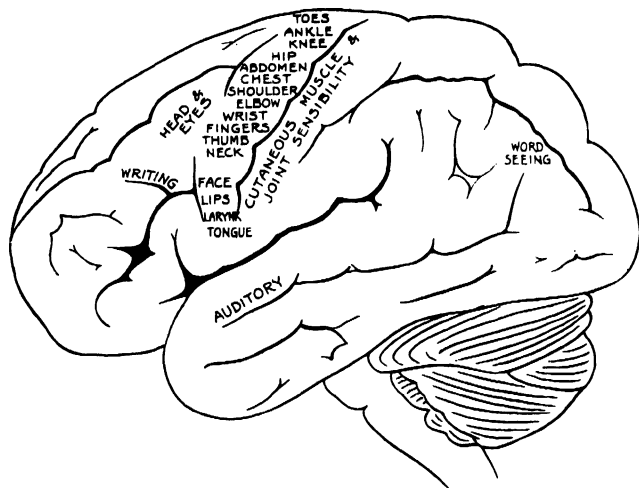


Fig. 64.—Localization in human cerebral cortex.

and of sense of position on the opposite side of the body, whereas pain sensation is affected only when the lesion involves the thalamus.

Leaving the afferent cortical and thalamic paths, we have yet another mechanism to consider. It has been noted that the afferent neurones conducting postural impulses form synapses with many other neurones before they terminate in connexion with the headwards-conducting neurones of the posterior column. One group of cells with which they are in communication lies in the mesial portion of the posterior horn, and is known as Clarke's column. From this group arises a tract conducting postural impulses headwards to the main organs for co-ordinating postural tonus, that is the cerebellum. The spinal afferent impulses therefore are conducted upwards, after having established their specific spinal reflex arcs, to three great co-ordinating mechanisms: to the cortex, which initiates purposive and discriminating movements; to the thalamus, governing vis-

ceral, motor, and secretory end-effects; and to the cerebellum, regulating postural tonus.

The cortical afferent system (Figs. 64, 65).—We have seen that the final path of the spinal afferent impulses is directed to the cerebrum, and it is here that they are brought into relation with the afferent impulses arriving from other receptors which are influenced by disturbances originating at a distance from the body, and which act on it through the intervening space by chemical (olfactory), radiant (visual), and physical (auditory) transmission.

The visual system (PLATE 22).—The visual impulses run backwards along the optic nerve; a partial decussation takes place at the chiasma, so that the fibres from the left halves of both retinae, which subtend the right halves of the visual fields, run together in the left optic tract, and vice versa. The fibres of each optic tract run backwards to the primary optic centres—the posterior part of the optic thalamus, the external geniculate body, and the superior corpus quadrigeminum. From the external geniculate body

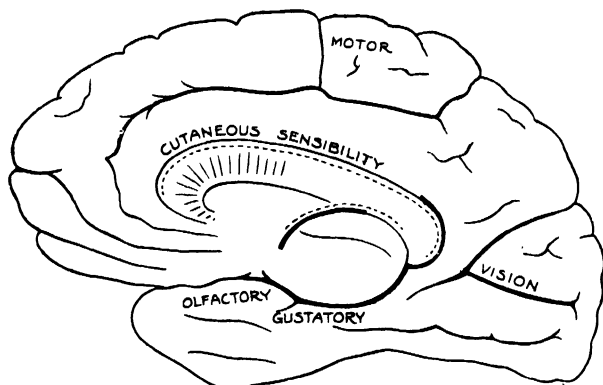


Fig. 65.—Mesial view of cerebral cortex.

and the optic thalamus new fibres convey the visual impulses to the cortical visual area. This area lies mainly on the mesial aspect of the hemisphere, being divided into an upper and lower part by the calcarine fissure which forms a boundary line between the cortical representations of the upper and lower quadrants of the corresponding half of the visual field. The afferent visual impulses from the retina conveying stimuli to the efferent pupillary neurones make their way directly to the corpora quadrigemina, whence communicating neurones continue a relatively short reflex arc to the efferent pupillo-motor neurones. The impulses which make their way ultimately to the visual cortex also form connexions, in the lateral geniculate body and the optic thalamus, with reflex arcs which, though infinitely more complicated than those of the spinal reflexes, are similarly characterized by a restricted field of reference. By means of these subordinate mechanisms an animal deprived of its cerebral cortex will avoid obstacles placed in its field of vision and will follow moving objects with its eyes, but if food is offered to it, or a whip is held up to it, it does not turn towards the food or away from the whip. The appropriate reaction to these latter stimuli involves the utilization of reflex paths of infinite complexity. It is from the visual area in the cerebral cortex that the intermediate neurones run which correlate the visual impulses with all such mechanisms as will by their interaction cause the dog to flee from the whip or turn to food—that is, to perform what we have termed a discriminating reflex. It is obvious that the reaction of these nervous mechanisms to the visual image of a whip is a result of stimuli that have occurred long before; it is this latent potential excitability as a result of preceding stimuli that is the physiological equivalent to the psychological event termed memory. In order, however, for a stimulus to become effective in a particular fashion, it is necessary for the nervous system to be attuned previously so as to react in one fashion and not in another to that particular stimulus. Take, for example, the auditory stimulus contained in the word “means.” A man says to me, “Wind means rain,” and I stretch out my hand for an umbrella; or he says, “Pluie means rain,” and I refrain from opening a French dictionary. The reaction to the auditory stimulus of the word “means” is determined by the nature of the latent excitability of the brain as a whole aroused by the preceding stimulus. It

is impossible to regard visual reflex memories as stored up as such in the visual cortex; the whole subject of cortical localization, as formerly stated, conveys an entirely erroneous impression. Certain areas of the cortex receive afferent impulses from specific groups of receptors, and if these areas be destroyed the redistribution of the impulses to other reflex mechanisms is rendered impossible; but a discriminating reflex, with its concomitant psychical equivalent of perception, cannot be a function of a single cortical centre any more than a simple spinal reflex can take place without the involvement of other reflex arcs. From the physiological point of view there is nothing to be gained by an attempt to detail the paths of connexion between the visual area of the cortex and other cortical mechanisms. It may be broadly stated that any cortical reflex process into which a visual nexus can be introduced, either by observation or the introspective study of its psychological concomitant, must be in communication with the cortical terminus of the visual fibres. In right-handed people the cortical area where visual impressions are correlated with the tactile and muscular mechanisms necessary for the movements of writing is localized in the left angular gyrus, and since the process of reading is in many people associated through the latent memory of muscular movements performed in writing, a lesion involving this centre may lead to word-blindness.

The auditory system.—The auditory impulses are conducted by paths which, like the visual, are in relation with centres through which complex reflexes of a non-discriminating nature take place, as the corpus geniculatum medium; they, too, finally terminate in the cortical auditory centre situated in the superior temporal convolution, where complex intercommunication with other cortical mechanisms occurs. All that has been said of the general function of the visual cortex applies *mutatis mutandis* to the functions of the auditory cortex. In right-handed people there is a similar connexion between the muscular and spatial afferent mechanisms connected with speech and the auditory system, and consequently auditory aphasia may result from a left-sided cortical lesion.

Other cortical sensory systems.—The olfactory system is connected with the uncinate gyrus. Gustatory afferent impulses appear to terminate in the cortex in the front part of the temporal lobe.

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The efferent path from the higher centres.—The cerebral integration of the various afferent paths has been briefly sketched and the efferent mechanism must now be considered. From the cerebral cortex run the efferent neurones which conduct to the motor terminal neurones in the spinal cord the efferent impulses of the cerebral reflex. To our ignorance of the terms in which the various afferent cerebral mechanisms inter-react must be added lack of knowledge of how the final integration of their interaction reaches that portion of the cortex whence the efferent cortical neurones arise. Experimental methods and clinical evidence have shown that in the precentral region of the cortex movements of the skeletal musculature of the opposite side of the body are represented. Stimulation of specific portions of this area will evoke definite co-ordinated actions, but isolated muscles cannot be caused to contract. The actions represented in the upper extremity of the precentral gyrus move the leg, those in the lowest part move the tongue and mouth, and the sequence of representation, followed downwards, runs: perineum, foot, knee, hip, abdomen, chest, shoulder, elbow, wrist, hand, eyelids, ear, nose, mouth, and tongue. The response of the individual points of the motor cortex is constant; thus, flexion of the arm is obtainable from one point, and extension from another. Strong excitation of any one may cause the successive involvement of the remainder of the motor cortex till the entire skeletal musculatures of one half of the body are affected.

The axones conveying efferent impulses from the motor cortex run directly to the motor neurones of the opposite side of the body. What has been said of the co-ordination of the muscles taking part in a spinal reflex applies equally here and need not be recapitulated. The contraction of antagonistic muscles is inhibited, and the action of the prime movers is steadied by synergic muscles. Certain actions can only be performed by the simultaneous contraction of both of a pair of muscles situated on either side of the body; thus, wrinkling the forehead involves the simultaneous contraction of both frontalis muscles, closing the jaw of both masseters, and retraction of the abdomen of both recti abdominis. Such actions are bilaterally represented in the motor cortex, and are only abolished by bilateral destruction of the motor cortex or of its efferent paths. The cerebral reflexes may reinforce and modify the spinal

reflexes when the spinal afferent stimuli which evoke the specific spinal reflex also pass headwards to the cerebrum; the voluntary movements that accommodate the body to, and aid the spinal reflex of, defaecation may serve as an example.

The postural mechanism.—In studying the spinal reflex we noted that, in addition to the end-effects produced by stimuli of the receptors by the environment, other reflex arcs tend to produce as an end-effect a tonic contraction of muscles designed to maintain the postural integrity of parts of the body, both when at rest and when liable to displacement as a result of active contraction of the skeletal musculature. The cerebral system constitutes a higher controlling mechanism for active movement, and the cerebellum is a similar system for the control and co-ordination of postural reactions. The impulses that reach the cerebellum arise from receptors not influenced by the external environment but by the conditions of internal stress existing in the muscles. These are co-ordinated in the cerebellum with stimuli generated in the vestibules. The maintenance of the postural stability of the body as a whole requires not only the co-ordination of the afferent impulses arriving from the muscles, but also those impulses excited by the angular and directional displacement of the body in relation to its environment which are conveyed by the vestibular nerve. The efferent paths of this mechanism govern the general tonic postural adjustment of the skeletal musculature. Destruction of one half of the cerebellum leads to a loss of tonus on the same side of the body, and to an inco-ordination in the execution of specific actions that are initiated by cerebral activity owing to the maladjustment of postural tonus. In the cerebral integration of the various afferent impulses that evoke a cerebral response the afferent impulses representing the position of the body in space must necessarily play a part, and hence the cerebellum is connected with the cerebral cortex and optic thalamus by afferent tracts.

The emotive mechanism.—Afferent impulses of painful and thermal nature, arising from stimuli potentially destructive to the organism, were traced upwards to the optic thalamus; here they are connected with an efferent mechanism tending to evoke generalized protective end-effects. Those end-effects which are manifested by the vaso-motor disturbances, deep respiration, the emptying

of the bladder and rectum, and the internal secretion of various hormones which influence autonomic structures, are termed emotive responses, and are of a protective nature, preparing the organism for action, be it fight or flight. Thus the startled bird defecates to secure the maximum lightness as it ascends in the air, and for a similar reason the fox vomits when bolted; and the hypersecretion of the adrenals raises the blood-pressure and mobilizes glycogen as sugar preparatory to intense muscular action. The chief efferent emotive impulses from the thalamus are directed ultimately to the efferent neurones of the vegetative nervous system by the thalamo-spinal paths.

In this brief exposition the nervous system has been considered from a purely physiological point of view, and the general functional integration has been sketched in general terms without entering into anatomical details. For more detailed information the reader is advised to consult the following works, viz.: "The Integration of the Nervous System," Sherrington; "Système Nerveuse Centrale," Soury; and "The Anatomy of the Nervous System," Edinger (English translation).

F. L. GOLLA.

NETTLE RASH (see URTICARIA).

NEURALGIA.—The term neuralgia, or nerve pain, is generally used to designate a paroxysmal pain whose source of origin is obscure, or pain which is referred over a much larger area than is supplied by the nerve in the neighbourhood of the irritant lesion. An instance of the latter is seen in cases of dental caries, in which an infected pulp-cavity in the lower jaw may set up pain not only in that gum but also referred pain in the upper jaw and cheek, and indeed over the whole fifth-nerve area on that side, and down the side of the neck to the clavicle. This spreading neuralgia is probably due to afferent painful stimuli setting up a hyperæsthetic state in the ganglion, and thus referred pain along its other branches. But spreading neuralgia is not always limited to the territory supplied by any one posterior root ganglion, since referred pains may be felt over a much wider area on the same side supplied by totally different nerves; for instance, an antral abscess before operation may cause a neuralgia not only over the second division of the fifth but over the whole trigeminal area on that side, and later

down the neck and arm even to the fingertips. The converse of this may be seen in some cases of trigeminal neuralgia when a tap upon the arm or on the same side of the body may start a spasm of pain in the face. Although the pain may be referred from one area to another in neuralgia due to some definite lesion that irritates a nerve, such as an infected pulp-cavity, it never crosses the middle line to the opposite side of the body. When what may be called "neuralgic pains" are bilateral they are due either to bilateral neuritis or to a central cord lesion, or else the pain is mental in origin, what is called psychalgia.

VISCERAL NEURALGIA

Referred pain from disease affecting various viscera, such as the stomach, spleen, kidney, ovary, prostate, heart and lungs, and eyeball, has been explained by Head, who showed how hyperæsthesia of definite posterior root areas on the skin of the trunk or head may be associated with a disease affecting organs whose sensory nerve supply is derived from the same spinal segment. In this way the stomach may refer on to the seventh, eighth, and ninth dorsal areas on the left side. Similarly the heart will refer from the sixth dorsal area upwards, pain due to aortitis causing hyperæsthesia on the second dorsal area, whereas mitral and tricuspid disease will refer as low as the fifth and sixth. The reason of this is that in the development of the heart its aortic end is nearest the head, while the mitral and tricuspid valves develop near the sinus venosus or its caudal end.

Errors of refraction in the eye, as hypermetropia or astigmatism, through inducing ciliary spasm, cause referred headache over the eyebrow near the middle line; gastric ulcer may be associated with areas of hyperæsthesia on the seventh to the ninth dorsal areas, according to whether the ulcer is cardiac or pyloric; the kidney and testes refer to the tenth dorsal areas of their own side, and so on.

Moreover, visceral neuralgia may be referred on to the scalp, through the agencies of the vagus nerve and its contiguity to the spinal root of the fifth nerve. Thus, gastric disorders may cause hyperæsthesia and headache in the left fronto-temporal region above the pinna—a form of headache familiar to many people after hastily swallowing a quantity of ice cream.

Functional pains may be associated in women with disease of the pelvic organs; ovarian

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neuralgia with tenderness of the tenth and eleventh dorsal areas is not uncommon, though care must be taken to exclude organic lesions, particularly appendicitis.

Neuralgic pains in the lower part of the back and sides are occasionally met with in young women of neurotic type. They are often worse at the periods, and are apt to be mistaken for rheumatism, fibrositis, sacro-iliac disease, sciatica, and other organic lesions; indeed, it is extremely difficult without prolonged observation of such cases to determine their functional origin. Similar pains round the neck and shoulders may be mistaken for brachial neuritis, osteo-arthritis, fibrositis, or cervical rib. Strictly unilateral pains are less likely to be functional in origin, particularly if certain movements regularly aggravate them, or if tenderness is constant at one or more points.

A fairly common form of functional neuralgic pain in the region of the lower end of the sacrum and coccyx, known as *coccygodynia*, is met with in both sexes, but more often in women. It is often ascribed to some insufficient cause, such as a slight fall, a diagnosis of dislocated coccyx or ruptured ligaments being not infrequently made; and excision of the coccyx has often been done for this condition, but by no means always with happy results. The possibility of this pain or paræsthesia in the coccygeal region being a psychalgia and mental in origin should not be overlooked, as it was in the case of a man who, fifty years ago, as a boy fell in a sitting posture on some stone steps. Ever since at intervals he has been suffering from pain in the region of the coccyx, though nothing locally wrong can be detected; the man himself, however, is notably neurasthenic and full of obsessions.

Lightning pains of tabes are sometimes referred to as tabetic neuralgia, though this is scarcely a legitimate use of the term. But in every case of persistent neuralgic pains in the limb or trunk it is advisable to examine the knee and Achilles jerks and the reaction of the pupils to light. Boring pain in the epigastrium may be associated with functional dyspepsia, though the importance of excluding gastric or duodenal ulcer, gastric cancer, spinal tumour, and caries, must not be overlooked. There appear to be rare cases of so-called crises of pain referred to the epigastrium or hepatic region and recurring at irregular intervals for years, in which the most careful search can detect no cause, such as tabes dorsalis, peptic ulcer, gall-stones, or appendicitis, and it

seems justifiable to assume that some such cases may be functional in origin.

CRANIAL NEURALGIA

Occipital neuralgia.—This is an uncommon neuralgia, and is generally bilateral. The pain is usually most acute about the level of the occipital protuberance, though a very tender point is common about half an inch below this level and about one inch from the middle line, over the point where the great occipital nerve pierces the deep fascia. The pain spreads over the back and sides of the head, sometimes as far as the vertex and mastoid region, and the scalp is liable to be hyperæsthetic and intolerant of the use of the hairbrush. Movements of the neck and head, sneezing and coughing particularly, are apt to cause exacerbations of pain. Occipital neuralgia may be due to osteo-arthritis between the atlas and axis vertebra, or to cervical pachymeningitis, malignant disease of the cervical spine, rheumatic fibrositis of the cervical muscles, or gouty, diabetic, malarial, and other forms of neuritis. Cold wind particularly may bring on or increase the neuralgia. **Treatment** by the application of dry heat is the most useful on the whole. Massage is to be avoided in these cases, and alcohol injection is not to be recommended. Treatment should be directed to the causes underlying the neuralgia.

Otic neuralgia.—The fact that some sensory cutaneous fibres are present in the seventh or facial nerve is the explanation of a somewhat uncommon form of neuralgia which at times may be of considerable severity in the region of the ear and auditory meatus. Usually associated with an outbreak of herpes on the tympanum and posterior wall of the auditory meatus, and sometimes in the cleft between the pinna and scalp, the pain is felt deep in the ear and radiates over the whole auricle and the side of the head above the pinna. In its most severe stage the pain may spread over the whole of the distribution of the trigeminal nerve and also into the back of the neck. The pathology of this condition is probably a herpes or a hæmorrhagic neuritis of the geniculate ganglion, and consequently it may be accompanied by facial paralysis; or loss of taste on one side of the tongue, due to involvement of the chorda tympani, may occur without facial paralysis, and may last several weeks or even months. The pain behaves like other forms of post-herpetic neuralgia, the paroxysms gradually subsiding in intensity,

and the hyperæsthesia of the ear and side of head gradually diminishing or leaving behind a numb or parchment-like feeling of the ear which may persist for many months. As in other forms of post-herpetic neuralgia, there is a liability in old persons for the pain to become chronic. **Treatment** should be by rest and sedatives, and later general tonic treatment; syringing the ear or other active interference with the tympanum is strongly to be deprecated.

Supra-orbital neuralgia.—This is a fairly common variety, and may be due to many causes, such as rheumatic neuritis or exposure to cold, or to post-febrile neuritis, following influenza, malaria, typhoid, etc. True supra-orbital neuralgia is nearly always unilateral, and the pain radiates from the supra-orbital notch over the eyebrow and forehead as far as the vertex. It is apt to be paroxysmal, and, when post-febrile in origin, may appear daily about the same hour, 10 or 11 A.M., and last for five or six hours. It is often excruciating in severity, completely incapacitating the patient while it lasts, and injection of morphia or heroin may be the only means of relief. In other cases a blow or an injury to the top of the head or forehead may be the origin of the pain, which varies in severity and is apt to be aggravated by cold winds, or when the patient is run down in health. Organic causes of this type of neuralgia may be cured completely by alcoholic injection of the supra-orbital nerve at the notch, or, if that fails, by resection of the nerve.

A common form of pain which may be mistaken for supra-orbital neuralgia is *migrainous neuralgia*, a form of migraine in which the nausea and vomiting are usually slight, or suppressed altogether. The pain is periodic, usually lasting many hours or even as much as two days, and is almost invariably associated with pain or intense tenderness on pressure on the temple, just in front of the hair line. Migrainous neuralgia may be completely unilateral, and in some cases affects the one side exclusively; but in the majority of sufferers the pain may alternate in different attacks, though it is always more severe on one side than on the other. In the history of these cases the paroxysmal attacks can often be traced back to early childhood, when they were regarded as bilious or sick headaches.

Other causes of pain which may simulate supra-orbital neuralgia are frontal sinusitis and the brow-ache associated with errors of

refraction such as hypermetropia and astigmatism.

Chronic pain referred over the distribution of the **superior maxillary** or the **inferior dental nerve** is occasionally met with, the pain being strictly unilateral, yet differing from true trigeminal neuralgia in the fact that it is continuous and not paroxysmal. Usually there is some hyperæsthesia of the skin in the distribution of the affected nerve, but movements of the jaw such as eating and talking do not increase the pain. The most rigid attention to the teeth fails to effect improvement, and antral abscess or other sources of peripheral irritation may be excluded. Chronic pains of this description have been, in my experience, limited to women usually in the early twenties, and a functional origin of the pain is an easy and seductive diagnosis. Yet treatment on functional lines is invariably a failure, while successful destruction of the nerve by operation or alcoholic injection arrests the pain entirely until regeneration of the nerve occurs. Possibly the real cause of the pain is chronic osteitis of the maxilla or mandible.

Chronic paroxysmal trigeminal neuralgia.—This affection, known also as *tiedouloureux*, *prosopalgia*, *trifacial neuralgia*, and *neuralgia quinti major*, is an inveterate paroxysmal pain affecting one or other side of the face in the distribution of the inferior or superior maxillary nerve, in many cases both of these being involved. But rarely is the supra-orbital branch of the ophthalmic division of the fifth involved, and then only when there is neuralgia of the second division also. In a few cases all three divisions of the nerve may suffer at once. The disease commonly starts about the age of 50, though wide variations in both directions are met with. About two-thirds of the sufferers are women, and in two-thirds of the cases the pain affects the right side of the face. True trigeminal neuralgia is seldom bilateral. Heredity of the actual disease is sometimes seen; in some cases blows upon the jaw or side of the head, in others exposure to cold wind, and in others again intense emotion, may appear as exciting causes of its onset. In many the pain begins after severe dental operations, though in others the sufferer may have been edentulous for years before the commencement of the neuralgia.

Symptomatology.—Absolute suddenness of onset of the initial paroxysm is a common feature of the disease, though intervals of complete remission of the pain, sometimes for many

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years, are frequently met with in the early history of a case. Sooner or later, however, the paroxysms return, usually with increasing severity, and the intervals of freedom are shorter. The most striking characteristic of this form of neuralgia is the complete absence of all pain or tenderness between the paroxysms, though these may recur as frequently as every three minutes during the day. Usually the sufferer is free at night, although this is not invariable. During the bouts of pain there is often intense hypersensitiveness of the skin, lips, side of nose or cheek, the lightest touch or breath of air being liable to start the attack. Some patients rock themselves and scream with pain, others press their hand on the affected side and are speechless during the spasm, in others the jaw may be fixed wide open, and there is usually an appearance of intense suffering. Saliva or tears may flow profusely, flushing of the cheek is common, and often some swelling of the side of the face is noticeable. Although there is so often great hypersensitiveness of the skin during the bouts of pain, there is no true hyperæsthesia, nor is there any anæsthesia. In neuralgia of the second division the pain is sometimes limited to the skin of the cheek, side of nose, and upper lip, and may not affect the upper jaw at all. In such cases treatment of the infra-orbital nerve by alcoholic injection in the infra-orbital foramen may arrest the neuralgia completely, perhaps for as long as four years or more. In the majority of cases, however, the pain is referred also to the gum and along the temporomalar branch, and often over the eyebrow. Infra-orbital injection will then be insufficient, and the nerve will require treatment farther back at its exit from the skull at the foramen rotundum. Neuralgia of the third division affects mostly the inferior dental nerve, but in these cases injection of the cutaneous branch at its exit from the mental foramen is very rarely of any value. Sometimes the pain is referred over the auriculo-temporal area, and sometimes the lingual nerve is involved, causing intense pain in the side of the tongue. When the paroxysms are severe and frequent, nutrition may be seriously interfered with, owing to the difficulty of taking food; indeed, the only way possible may be to suck liquid nourishment through a straw placed in the opposite corner of the mouth. For speech, writing has to be substituted. The pain is usually described as darting and quivering, or as resembling red-hot knives or Bradaws

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thrust into the cheek; it often ends like an explosion of fireworks in the face. The origin of these neuralgias has been considered mysterious; briefly it may be said that no disease can be found in the gasserian ganglion or nerve-trunks. Arterio-sclerosis has certainly nothing to do with it. Possibly septic neuritis of the dental nerve filaments associated with a chronic infective osteitis is the source of the reflex neuralgia.

Treatment is mainly operative. Drugs have little or no value, though tincture of gelsemium in large doses occasionally helps. Neurectomies are inadvisable, and the choice lies practically between gasserectomy and alcoholic injection of the nerve trunks. The latter, when done properly, often confers complete relief for two or three years, or even longer, and re-injection can be done on the return of the pain. In many cases it is possible to destroy the gasserian ganglion itself by alcoholic injection, in which case cure is likely to be just as permanent as after gasserectomy.

WILFRED HARRIS.

NEURALGIA, DENTAL (see DENTAL PAIN).

NEURASTHENIA.—A functional disorder, resulting from exhaustion of the nervous system, and probably of certain endocrine glands, especially the suprarenal, which manifests itself by abnormal mental and physical fatigability, and irritability of the nervous system.

Etiology.—Neurasthenia may occur at all ages, but is rare before puberty. Males and females are equally affected. It results most frequently from a combination of mental and physical fatigue. Mental overwork rarely leads to neurasthenia until sleep is affected and physical fatigue is thereby induced. It is particularly likely to cause symptoms if associated with worry. Eyestrain, caused by errors of refraction, especially astigmatism and weakness of convergence, is a frequent accessory factor. Uncomplicated physical fatigue, due to excessive exercise with insufficient rest, leads to symptoms of exhaustion, but these hardly amount to neurasthenia unless some other etiological factor is present. Repeated pregnancies and prolonged lactation frequently lead to neurasthenia, especially among the poorer classes. The influence of accidents is considered in NEUROSES, TRAUMATIC.

Toxæmia in many cases plays an important

part. Neurasthenia is frequently a result of acute fevers, especially influenza, and, in hot climates, of malaria, typhoid fever, and dysentery, as well as of such chronic infections as pyorrhœa alveolaris, sinus disease, and chronic appendicitis. The importance of intestinal toxæmia has been greatly exaggerated; diarrhœa is much more prone to give rise to neurasthenia than constipation, unless the latter is treated by the excessive use of aperients, as toxins may be absorbed from fluid feces, but not from the solid scybala present in the colon and rectum in most cases of constipation. Alcoholism predisposes to neurasthenia, but can hardly be regarded as an actual cause. Exposure to excessive heat is an important factor, and is in part responsible for the exhaustion which is often attributed solely to fevers contracted in the tropics. Sexual excess of any kind is undoubtedly a factor, but there is no evidence that masturbation *per se* is of any importance.

Pathogenesis.—Many of the etiological factors just described have been shown experimentally and by post-mortem examinations in man to result in exhaustion changes in the nerve-cells of the brain and in the cells of the suprarenal glands and the liver. It is these changes which cause the main symptoms of neurasthenia, and the latter can be roughly classified as due either to brain or suprarenal exhaustion. Rest relieves the symptoms by promoting regeneration of the affected cells. Neurasthenia has to this extent an organic basis, and thus differs from other functional nervous disorders in not being of purely psychical origin. The neurasthenia of the climacteric is largely due to hormonal deficiency: the ovaries are primarily concerned, but from a therapeutic point of view the thyroid insufficiency is much more important.

Symptomatology.—The most important and commonest symptom is insomnia, as it aggravates all the other symptoms, and so long as it persists recovery is impossible. The patient generally finds great difficulty in falling asleep, and when at last sleep comes it is often so light that the least sound awakens him. Instead of settling to sleep again he is so worried about his insomnia that he becomes wide awake and spends the rest of the night tossing about, and when it is time to get up he feels more tired than before he fell asleep. If a healthy man has a bad night his head feels heavy in the morning; mental or physical work leads to rapid fatigue, and he

finds difficulty in concentrating. If the insomnia is repeated every night for a week these symptoms become more and more pronounced. It is thus only natural that the insomnia of the neurasthenic should also lead to these symptoms. They are in part due to the same nervous exhaustion which causes the insomnia, but they are always aggravated by the latter, and in chronic cases they are often due solely to the insomnia and disappear entirely as soon as sound sleep returns. The headache is rarely severe. Most commonly the patient complains of an uncomfortable heavy sensation, which is often worst in the morning, and gradually improves towards evening. He has no inclination for activity of any kind in the early part of the day, and if he has mental or physical work to do the effort required to accomplish it is considerable, and fatigue rapidly occurs. He is incapable of sustained mental activity, and cannot concentrate his thoughts on his business or even on light reading. His mind is clearest in the evening, and he may then be able to take some exercise without undue fatigue.

The excessive physical fatigability results in aching of the limbs, and still more frequently of the back, which is only relieved on lying down; it makes every muscular effort difficult. The fatigue is often increased by the patient's inability to relax his muscles completely, even when he is in bed and trying to rest. A fine tremor of the hands is frequently observed when they are held out, but this is secondary to the rigidity, and disappears as soon as the patient is taught to relax his muscles. Weakness of the abdominal muscles leads to visceroptosis, which aggravates the abdominal discomfort, the general sense of fatigue, and the circulatory insufficiency; but the abnormal mobility of the right kidney, which is very common, especially if much weight has been lost, does not give rise to any symptoms unless the patient unfortunately becomes aware of its existence. The tendency to muscular fatigue affects the eye muscles, so that accommodation and convergence become increasingly difficult, and the patient cannot read for any length of time without making his headache much worse.

The patient feels so weary and miserable in the early morning that he has no appetite for breakfast. In severe cases the anorexia persists through the day, but in slighter cases he may enjoy his midday and especially his evening meal. Owing to the anorexia too little

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food is taken, and the patient loses weight and becomes still more exhausted. The bowels do not receive the normal stimulus afforded by an adequate diet, and constipation results. The patient, already anxious about his health, worries still more about this, and doses himself with purgatives, which only aggravate his condition by irritating his bowels, causing abdominal discomfort, and producing an artificial diarrhoea. Visceral reflexes tend to be exaggerated owing to the irritability of the nervous system; consequently, intestinal spasm and excessive secretion of mucus may occur, the condition known as muco-membranous colitis resulting, but this rarely causes much trouble till the patient discovers the mucus. The irritability of the exhausted nervous system also leads to an increased sensibility to visceral sensations, and the patient becomes aware of the activity of processes which should pass unnoticed. If for any reason his attention is drawn to his abdomen, his digestion, which is not normally felt, now falls within the range of consciousness, and he complains of fullness and discomfort after meals. Radiographic investigations have proved that atonic dilatation of the stomach, which has often been described as very common in neurasthenia, is comparatively rare, and that the digestive symptoms are independent of any disturbance in the motor functions of the stomach. The secretory functions also show no constant alteration. The patient generally reduces his diet in the hope of losing his discomfort; this results in secondary anorexia, and the result is that his weight becomes more and more subnormal.

In other cases the patient's attention is drawn to his circulation. Any slight inefficiency caused by the same combination of mental and physical overwork with the toxæmia that led to the exhaustion of the nervous system, gives rise to symptoms which would be ignored by a man with a normal nervous system, but which become a great source of worry to the neurasthenic. He complains of palpitation, præcordial discomfort, shortness of breath on exertion—exactly the symptoms to which the label "D.A.H." or "disordered action of the heart" has been attached in soldiers. These symptoms are really independent of any cardiac disorder, and are a part of the neurasthenia; they disappear with the latter, especially if the patient is taught to disregard them and to realize that they do not indicate heart disease.

If the suprarenal factor is prominent, especially when the neurasthenia is a sequel of dysentery, malaria, or enteric fever, or of an exceptional degree of emotional disturbance and physical exhaustion, the blood-pressure is subnormal, digestive symptoms are prominent, and even pigmentation of the skin may be present. The clinical picture may then closely simulate that of Addison's disease, but with suitable treatment rapid improvement and finally complete recovery occur, as the suprarenal insufficiency is due to exhaustion of the gland and not to organic disease.

Neurasthenia often leads to deficient sexual activity, and this too is a very great source of worry, especially to men. The more the patient thinks about it the worse he becomes, and complete functional impotence may result. At the same time he is likely to complain of neuralgic pain in his testicles or perineum, and a varicocele, though of no real importance, becomes a source of much apprehension.

With his attention drawn to his digestion, his circulation, or his sexual functions, the neurasthenic becomes increasingly hypochondriacal, and prepares detailed descriptions of his symptoms and long lists of questions to ask his doctor. He is then very liable to develop symptoms of psychasthenia (q.v.). But if he is made to understand the true significance of his symptoms, and that they indicate no organic disease, they will pass away completely as he recovers from the neurasthenia to which they are secondary.

Diagnosis.—A thorough physical examination is essential before neurasthenia can be diagnosed, as it is necessary to exclude all organic debilitating diseases which may give rise to similar symptoms. The most important of these is *tuberculosis*, the general symptoms of which in the earlier stages are simply those of neurasthenia. The *anemias* and *chronic Bright's disease* can be excluded by examination of the blood and urine respectively. Certain organic nervous diseases may at first simulate neurasthenia, but in the latter there are no abnormal physical signs. If there is the least possibility of *general paralysis*, which in the earliest stage may closely simulate neurasthenia, the cerebro-spinal fluid should be examined for cells and for the Wassermann reaction.

Prognosis.—Neurasthenia is a curable condition, the rapidity of the cure varying inversely with the duration of symptoms. The success of treatment largely depends upon the

patient's home and financial conditions, for if his worries are irremovable or recurrent, a permanent cure can hardly be expected. Neurasthenic patients are often abnormally suggestible, and are consequently specially liable to develop hysteria; psychasthenia is also a very common complication. The presence of the former does not greatly affect the progress of the case, as it is generally easily dealt with, but the latter may require prolonged treatment before recovery takes place. Neurasthenia never leads to insanity, but the inability to concentrate and the rapid mental fatigue often lead to the fear of insanity, which should at once be dispelled, as otherwise it is likely to result in superadded psychasthenia.

Prophylaxis.—It is doubtful whether a neuropathic family history predisposes to neurasthenia, so that the question of prophylaxis in childhood hardly arises. It is, however, the duty of every doctor to point out to those of his patients who lead a strenuous life how necessary to health are intervals of rest. No man can do hard mental or physical work on more than six days a week, or for more than eleven months in the year, without breaking down sooner or later. Adequate holidays—real holidays, in which a man cuts himself completely away from his work—are the best preventive of neurasthenia.

Treatment.—The two essentials for the successful treatment of neurasthenia are, firstly, removal of the cause, if it is still operative, and, secondly, the placing of the patient in a condition which will as rapidly as possible lead to the recovery of his nervous system from its exhaustion. As the most important cause is mental and physical overwork, indicating the need of rest, and as rest is also the chief means of restoring the exhausted nervous system to a healthy condition, this will be the first method considered.

In severe cases, especially where much weight has been lost, complete rest in bed is required, but the old-fashioned rest cure of several weeks' duration is rarely, if ever, indicated. I have known many women who have become lazy hypochondriacs for the rest of their lives as the result of a too thorough Weir Mitchell "cure." A week or a fortnight in bed is generally enough, but from the start the patient should walk to his bath and to the lavatory, so as not to let his legs become too weak from disuse. When the complete rest is finished, he should get up for half an hour

in the morning and afternoon, increasing the period by half an hour every day until he only rests for an hour in the afternoon. At the same time he should train himself to take more and more exercise, beginning with a quarter of an hour's walk half an hour before lunch, tea, and dinner, and gradually increasing the duration and strenuousness of the exercise.

The majority of neurasthenics lie more or less rigidly in bed, often with the head raised from the pillows and their arms off the bed. The patient should be made to rest in such a way that he feels as if he were falling through the bed, not as if he were holding up the bed by his own muscular effort. When the limbs are raised from the bed and dropped they should fall quite passively, and the same thing should occur when the head is lifted off the pillow.

In mild cases a holiday away from home is all that is required. A man must get away from his business or profession, leaving his affairs in such a state that he is satisfied they will work smoothly in his absence, so that complete separation from his ordinary occupation will not worry him. A woman, whose chief daily occupation consists in looking after her home and family, must go to some place where she has no household cares, and it is generally essential that she should leave her children at home. It is often desirable for the patient to stay in bed for breakfast and only to get up at 11 or 12 o'clock. In every case he should rest for an hour in the afternoon, and, if possible, should sleep. He should be made to realize that he will not endanger his night's rest by doing this, as many people who sleep badly at night and purposely avoid sleeping during the day, however tired they may feel, erroneously think. The rest of the day should be spent in congenial diversions, but it is most important that these should not be so strenuous or prolonged as to cause more than a pleasant sensation of slight fatigue. Outdoor occupations, such as walking, bowls, and croquet, and later golf, fishing, and riding, are the most satisfactory, and the patient should gradually train himself by making a small but definite addition to the duration of his exercise every day until he is in first-class physical condition. He should avoid reading or writing for any long period at a time, and his reading should be light in character. Eyestrain should be corrected at once by the provision of suitable glasses. In some cases in which the strain is in part due to asthenopia, the patient should

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not be allowed to read, write, or do needlework until considerable improvement has taken place.

Complete sexual abstinence should be enjoined at first, especially when sexual symptoms have been present, and the patient should be made to understand that any troubles of this kind he has had in the past will disappear as the result of his rest.

The treatment is most satisfactorily carried out in the country, where the patient is not disturbed by the noise of traffic; it is also likely to be much more beneficial away from home, unless he has a large country house and can be entirely relieved of all business and household worries. There is, however, no reason why he should not receive and write letters, so long as these are not of a business or worrying character. The complete isolation of the Weir-Mitchell treatment irritates the majority of neurasthenic patients, and is more likely to make them thoroughly unhappy than help them to get well.

It is of the utmost importance that the patient should sleep from the commencement of treatment. In some cases the mere removal from his home and business surroundings and the quiet of the country will result in a return of normal sleep. If this fails, simple psychotherapy should always be employed. The patient should be told that his insomnia is largely a bad habit, and that he sleeps badly because he expects to do so. If it has begun as a result of overwork or worry, he must be talked to until he feels quite convinced that he will sleep well directly he gets away. If he has had difficulty in falling asleep, this will disappear if he is really convinced. The effect of the conversation will often be increased if the patient is told to do two or three things which he believes may have a soporific effect; thus, he may have a very short but very hot bath just before getting into bed and then take a hot drink. Between dinner and going to bed he should occupy his mind in some unexciting fashion, which, however, should be of sufficient interest to keep his mind occupied with thoughts of it on getting to bed, to the exclusion of business or home worries. If he has found difficulty in falling asleep again after he has waked in the night, it should be explained to him that this is solely due to the fact that he has got into the habit of waking himself up. If he regains consciousness during the night, he should see to it that this is only a passing thing, and that he should not even open his eyes,

but after moving into a more comfortable position he should make up his mind to fall asleep again; if instead of this he opens his eyes, turns up the light to see what time it is, and perhaps has a drink or begins to read, simply because he has been in the habit of doing this, he will thoroughly wake himself up and may have great difficulty in getting to sleep again.

In severe cases of insomnia, suggestion under hypnosis is extremely satisfactory; it is, indeed, one of the very few conditions in which I believe that hypnotism is almost indispensable. It does not matter what time the patient is hypnotized, nor what method is used; very light hypnosis is all that is required. It is not the hypnotism itself that acts, but the suggestion of sleep, made when the patient is in a mental condition entirely uncritical and extremely prone to accept suggestions, that is so effective. He is told repeatedly that he will sleep well to-night, and indeed every night; that he will fall asleep directly he lies down and will go to sleep again at once if by any chance he wakes during the night; that the cause of his insomnia is gone, and he will therefore get perfectly well now. It is rare that the treatment requires repetition on more than three or four consecutive evenings. If it is not completely successful at the end of a week it should not be continued, for the hypnotism habit is just as undesirable as the drug habit.

Only in the rare cases in which hypnotism has failed, or in which for any reason it cannot be used, should drugs be given. The habitual use of hypnotics in neurasthenia does an enormous amount of harm. When drugs are required, the best combination is medicinal with acetyl-salicylic acid (aspirin); 7-15 gr. of the former, depending upon the severity of the case, should be given with 15 gr. of the latter. The same dose should be given the second night, but on the third night the dose of medicinal should be reduced by 1 gr. without altering that of the aspirin. The dose of medicinal is then further reduced by 1 gr. every other night until none is given. By this time the patient is almost invariably sleeping well. Such a "medicinal series" has a great advantage over the ordinary way of giving hypnotics, as the treatment will only last a definite number of nights—a maximum of thirty when the first dose is 15 gr.—and there is no danger of developing a drug habit.

Sources of chronic infection, such as pyor-

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rhœa alveolaris, septic tonsils, sinus disease, and chronic appendicitis, should be sought for and dealt with.

Patients without gastric symptoms, who have not lost weight, should make no change in their diet. If they have lost weight they should be encouraged to eat as much as possible, and given a glass of milk before breakfast, in the middle of the morning, at tea-time, and on going to bed. A thin patient, who cannot eat anything on account of anorexia, can be made to regain his appetite by explaining to him the need of food and how the appetite will return as soon as more is taken, and at the same time persuading him to eat more even if he finds it very difficult at first. Special dieting, particularly the use of large quantities of milk without any solid food, should be avoided. The numerous patent foods, and especially the powders containing mixtures of casein with phosphates and other chemicals, and the combination of wine with meat extracts, only benefit their manufacturers. If dyspeptic, the patient should be treated on the lines laid down under Neurasthenic Dyspepsia, in STOMACH, FUNCTIONAL DISORDERS OF.

Alcohol should only be allowed if it seems likely that its use will improve the appetite and help the patient to sleep, especially in very debilitated and elderly individuals. It should be taken with meals and in strictly regulated quantities, and the patient himself should be consulted as to what form of alcohol he likes best. Excessive smoking should be forbidden.

Constipation should be treated by diet and regulation of the habits. Drugs should be avoided if possible; if something must be given, the dose should be as small as possible. Massage has no special virtue in neurasthenia. When a patient is to be kept in bed for two or more weeks, massage for his limbs and back, given for not more than half an hour, helps to keep his muscles in good condition, and abdominal massage is useful if there is any dyspepsia or constipation, but no massage should be given after he has begun to get up for two or more hours a day, as it then only tends to make him hypochondriacal. A cold bath in the morning, or a cold douche after a hot bath of short duration, is beneficial from the beginning of treatment. After much experience in charge of an electrical department, I am quite convinced that no form of electricity is of any value in the treatment of neurasthenia. High-frequency and static elec-

tricity can only act by suggestion, and as the symptoms in neurasthenia are the physical result of exhaustion and are not hysterical and due to suggestion, gross methods of suggestion with elaborate electrical apparatus is contraindicated, if indeed it is ever a justifiable method of treatment.

I do not believe that any drugs are of real value in neurasthenia. So-called nerve tonics, such as hypophosphites, glycerophosphates, lecithin, formates, and "sérum neurasthénique," have all had their phases of popularity, but they are without action of any kind on the nervous system. Arsenic does not appear to be any more useful. A recent trial of valerian in various forms confirms my earlier impression of its complete futility. Strychnine in doses sufficiently large to have any appreciable effect increases the irritability of the already irritable nervous system; any stimulation it produces can only harm a nervous system which requires rest and not stimulation. Bromides have been largely used in the past for the associated psychasthenic symptoms, but any calming influence they may exert can be more efficiently exerted by psychotherapy. Possibly very small doses (5 gr. twice a day) for long periods given to patients with a congenitally irritable nervous system may be of value, but the evidence is still inconclusive. Animal extracts, such as the so-called lymph compound and tablets said to contain hormones extracted from a great variety of organs, are often inactive, and when active their use is grossly empirical, as the inter-relation of the various glands is such that when one acts deficiently others are overactive. As it is quite impossible in our present state of knowledge to say exactly which hormones are deficient in a given case, and in what doses each should be given, it is absurd to expect that a mixture of every obtainable hormone in fixed relative doses should ever be suitable. There is, moreover, no evidence that these products exert any effect apart from the suggestion which is present when the patient knows what they are supposed to contain and what they are expected to do, unless some really active substance such as thyroid happens to be present, in which case it is very likely that the dose will be excessive and do more harm than good.

A. F. HURST.

NEURASTHENIC DYSPEPSIA (see
STOMACH, FUNCTIONAL DISORDERS OF).

NEUROMA

NEURITIS, LOCAL (*see* SPINAL NERVES, LESIONS OF).

NEURITIS, MULTIPLE (*see* MULTIPLE NEURITIS).

NEURITIS, OPTIC (*see* OPTIC NEURITIS AND NEURO-RETINITIS).

NEUROMA.—The nerves may be the seat of tumours which are called neuromata. Of these there are two varieties, the true and the false. In a *true neuroma*, or **ganglion-neuroma**, nerve-fibres and ganglion cells form an essential part of the growth. These are exceedingly rare and are only found in connexion with the sympathetic system in the thoracic or abdominal cavities. The *false neuroma*, or **neuro-fibroma**, is much more common. It arises in the connective tissue of the nerve, and is fibrous, though it may undergo cystic or myxomatous degeneration. It may lie to the side of the nerve, or may be central and separate the bundles of the nerve-trunk. The nerve-fibres passing through such a fibrous growth are usually normal.

Neuro-fibroma may be solitary or multiple. When *multiple* the tumours may be confined to a single nerve, or to a plexus, or be widely spread throughout the spinal nerves and involve the cranial nerves also. They vary in size from small nodules a few millimetres in diameter to large masses several inches in length. Their origin is obscure. They may be congenital and are often associated with a neuro-pathic inheritance. They usually appear in early life, and the subject may be mentally defective or epileptic. *Solitary* neuromata more often produce symptoms than the other forms. These are generally severe intermittent pains which radiate distally along the nerve from the site of the growth. Objective motor or sensory disturbances in the distribution of the nerve are the exception.

Tubercula dolorosa are multiple tiny tumours lying beneath the skin on the terminal twigs of cutaneous nerves. They may be very tender and painful. In *general neuro-fibromatosis* there may be hundreds of tumours on the various nerves. These may be visible through the skin. They are soft and cyst-like in consistence, and they rarely give rise to nervous symptoms, but may be mechanically inconvenient, and if present on spinal or cranial nerve-roots may cause a compression paraplegia or the signs of intracranial tumour. Some auditory-nerve tumours are of this nature. When to

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this generalized neuro-fibromatosis are added pigmentation and numerous sessile and pedunculated tumours of the skin, the condition is known as *von Recklinghausen's disease*, or *molluscum fibrosum* (*see* SKIN, FIBROMATA OF). A rare condition is *plexiform neuroma*. This usually affects the trigeminal nerve, which is twisted and irregularly thickened so as to form a tumour-mass in the face.

All these forms are slowly progressive, but, unless pressure effects on the cord or brain are produced, the condition does not materially shorten life. Operation may be indicated for solitary growths or for such multiple tumours as cause inconvenience. Good results have been described in multiple fibromata from the use of injections of fibrolysin. Otherwise treatment must be purely symptomatic.

F. M. R. WALSH.

NEURO-FIBROMA (*see* NEUROMA; SKIN, FIBROMATA OF).

NEURO-MYOSITIS (*see* MYOSITIS).

NEURO-RETINITIS (*see* OPTIC NEURITIS AND NEURO-RETINITIS).

NEUROSES.—Many of the difficulties in the diagnosis and treatment of the neuroses would disappear if such terms as "functional," "neurosis," "neurasthenia," "hysteria," and "psychasthenia" were precisely defined, and were not used, as is too often the case, as if they were more or less synonymous.

A *neurosis* is a *functional disorder which depends upon abnormal action and reaction of the nervous system*. By a *functional disorder* is meant one which does not depend on any organic lesion, although it may depend upon toxæmia or abnormal hormonal action as well as upon abnormal nervous action. The word *psychoneurosis* should be reserved for the neuroses—hysteria and psychasthenia—which are psychical in origin and are distinct from those, such as asthma and migraine, which depend upon abnormal action at a lower level of the nervous system.

A *neurotic* individual is one who has an inborn or acquired abnormal liability to develop neuroses. The word *neuropathic* is really synonymous with neurotic, and has no advantage over it.

Hysteria, psychasthenia, and the sympathetic neuroses are the only pure psychoneuroses, which are psychical in origin and entirely functional, being dependent upon nothing else

than abnormal action of the nervous system. As they are of psychical origin, the rational treatment is psychotherapy, the psychical effect of a rest cure and of drugs, diet, and physiotherapy being alone of value in such cases.

1. **Hysteria** (q.v.) is a condition in which symptoms are present that have been produced by suggestion and are curable by psychotherapy.

2. **Psychasthenia** (q.v.) is a functional nervous disorder characterized by inability to co-ordinate the mental processes, which results in failure to regulate ideas and actions in a logical manner, together with difficulty in concentration and, in more severe cases, in obsessions and emotional crises.

An **obsession** is an irrational idea or unsubstantial fear, commonly called a phobia, which intrudes itself into consciousness in an irresistible manner without completely filling or dominating it.

Emotional crises are attacks of apparently causeless emotion, such as dread, horror, or terror, with their physical accompaniments of tremor, palpitation, sweating, and even diarrhoea, and of laughter or weeping, popularly called "hysterics."

3. **Neurasthenia** (q.v.), which is generally regarded as a neurosis, is a disorder resulting from exhaustion of the nervous system and probably of certain endocrine glands, especially the suprarenal, which manifests itself by liability to abnormal mental and physical fatigue and by irritability of the nervous system. But strictly speaking it is not a neurosis at all, as the exhaustion is associated with organic, though temporary, structural alterations in the nervous system and endocrine glands.

4. **Paroxysmal neuroses.**—Another group of diseases which can be correctly described as neuroses are those depending upon the existence of certain irritable centres in the nervous system, the irritability being generally congenital, but perhaps also sometimes acquired. Among these are asthma, hay fever, mucomembranous colitis, and migraine. In *asthma*, which may be taken as a type, the irritable centre is the part of the vagal nucleus in which the constrictor fibres to the bronchi arise. It may be called into activity by very minute doses of proteins contained in various pollens, in foods, in cat and horse dandruff, and in certain bacteria, to which the centre is specifically hypersensitive; by nervous impulses acting reflexly from the nose, throat,

bronchi, stomach, and intestines, as well as by purely psychical stimuli. The resulting spasm of the bronchi with the associated congestion and oversecretion of mucus constitutes an attack of asthma. Asthma thus depends upon abnormal action and reaction of the nervous system and not upon any organic lesion; it may therefore be regarded as a respiratory neurosis, although it has nothing to do with the psychoneuroses—hysteria and psychasthenia.

Such neuroses as asthma and migraine do not occur more frequently in neurotic than in normal individuals. They do not respond to psychotherapy unless, as sometimes happens, an hysterical element is superadded. The rational treatment is to reduce the irritability of the abnormal focus in the central nervous system and to remove the peripheral causes which bring it into action.

5. **Sympathetic neuroses.**—All emotions, but especially those of fear and anger, are accompanied by physical phenomena which depend upon the activation of the sympathetic nervous system by the emotion. When the painful memory of the event which gave rise to the emotions is repressed, or when the struggle between conflicting emotions remains unsolved, the primary mental symptoms of psychasthenia may be associated with some of these physical phenomena. This is the explanation of many of the visceral neuroses, which are commonly classed as "functional" or "nervous" on account of the absence of any sign of organic disease, but without any more definite information being vouchsafed as to their pathogenesis.

The chief sympathetic neuroses are gastrointestinal and circulatory.

(a) **Gastro-intestinal sympathetic neuroses.**—Emotional stimulation of the sympathetic nerve-fibres to the alimentary canal results in inhibition of the movements of the stomach and intestine, contraction of the pyloric and ileo-caecal sphincters, and inhibition of gastric, intestinal, and pancreatic secretion. Serious interference with digestion results, and various symptoms, commonly grouped together as nervous dyspepsia, develop.

(b) **Circulatory sympathetic neuroses.**—Sympathetic activity leads to a rise in the pulse-rate and in blood-pressure. Many cases of persistent tachycardia and persistent hypertension are of this nature. They can be recognized by the absence of evidence of organic

disease, and by the fall in pulse-rate and blood-pressure to normal which occurs under hypnosis and during natural sleep. The high blood-pressure, though nervous in origin, may lead to such serious organic sequels as cerebral hæmorrhage. The symptoms may also be intermittent in the form of crises of pseudo-angina and of paroxysmal tachycardia, sometimes accompanied by profuse sweating.

The futility of the ordinary treatment of these digestive and circulatory neuroses by bromides and valerian and by rest cures is obvious, but intelligent psychotherapy, which reveals the true origin of the symptoms to the patient and helps him to master his emotions, leads to gradual disappearance of the symptoms.

(c) **Hormonic neuroses.**—The suprarenal and thyroid glands are innervated by the sympathetic system, and a condition of continued or intermittent *functional hyperadrenalism-hyperthyroidism* may occur as the physical expression of the suppressed emotional disturbances of psychasthenia. The adrenalin reinforces the primary sympathetic activity by stimulating the receptor substance which exists between every sympathetic nerve-ending and the tissue it supplies. It is therefore impossible to differentiate the effects caused by primary sympathetic activity from those caused by the secondary activity of the suprarenals. The effect of thyroid hypersecretion is more obvious, as this stimulates mainly organs supplied by the cervical sympathetic, the chief symptoms being tachycardia and exophthalmos with retraction of the upper eyelid and dilatation of the pupil, the general metabolism being at the same time increased. The picture presented in functional hyperadrenalism-hyperthyroidism is therefore very similar to that of Graves's disease, which is an organic condition due to primary involvement of the thyroid gland, and not a neurosis; but the thyroid is not hypertrophied, any slight enlargement of the gland being temporary and due to the active congestion caused by increased functional activity; and the blood-pressure is raised instead of being normal. The symptoms vary greatly from time to time, the eyes becoming more prominent and the pulse more rapid with the slightest emotional excitement. The condition responds rapidly to rest and psychotherapy, but local treatment with X-rays and by operation, though it may be of great value in Graves's disease, is clearly contra-indicated.

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NEUROSES, OCCUPATION.—Although the term is not infrequently employed in a somewhat loose sense, an occupation neurosis is understood to signify a disturbance of motor function which occurs as a sequel to the constant repetition of a particular movement or series of movements, and is seen only when such movement or movements are attempted.

Etiology.—Occupation neuroses may occur in either sex and in any rank of society; they are seen probably with greatest frequency in young adults, though also, of course, in older people. There is little doubt that the presence of an occupation neurosis is an indication of an essentially neuropathic diathesis, for very many subjects who have in the course of their occupation to make constant repeated similar movements never develop an occupation neurosis. The condition is found associated with general neurasthenic symptoms in many instances, and it may be regarded as an expression of this general neurasthenic state. On the other hand, occupation neuroses may develop on a more definitely organic basis, e.g. actual neuritis, or arterio-sclerosis of the vasa nervorum, or in cases of otherwise latent cervical rib. Where an apparent organic basis is discoverable, the condition should be differentiated from what may be designated idiopathic occupation neurosis.

Varieties.—As the most common occupation is that of writing, so the commonest form is writer's cramp or scrivener's palsy. Other frequently-seen forms are those of the telegraphist, violinist, typist, pianist, cigarette-maker, hammerman, painter, plasterer, as also those of subjects who use their fingers for fine movements as in sewing and knitting. Occupation neuroses in the legs have been observed in turners, lathe-workers, sewing-machinists, and others who have to work a pedal or pedals with their feet. Players on wind instruments and glass blowers sometimes develop an occupation neurosis of facial muscles. Some forms of torticollis are essentially of the same nature.

Symptoms.—A workable classification of symptoms into spasmodic, paralytic, and tremulous has been made. It should be understood, however, that no sharp lines can be drawn between these types.

1. *Spasmodic.*—In this type the patient finds that when he essays certain movements a spasmodic cramp, sometimes a painful cramp, seizes the muscular group or groups involved. Thus, in writer's cramp he may involuntarily push the point of the pen through the paper,

or his hand may be lifted off the paper, or cramp-like fixation of the fingers may immobilize the pen or pencil. In violinist's cramp either the arm that is making the bowing movements may be seized with cramp, or the fingers of the other hand that are moving on the strings. Often abrupt, jerking movements effectually prevent all efforts at smooth, co-ordinated innervation.

2. Paralytic.—Sometimes there is no cramp, but a more or less sudden loss of power in the muscles concerned. In one case of pianist's cramp the patient was unable to make any movement with his right arm to beyond a certain position on the keyboard; there was simply cessation of innervation necessary for further abduction from the middle line. Sometimes the pen falls from the grasp, the bow from the overworked fingers. It is a sort of suspension of function from temporary exhaustion.

3. Tremulous.—The movements may be those of fine or coarse tremor or tremulousness. The reader must have noticed that some persons are prone to a somewhat unnecessary and showy to-and-fro movement of the wrist and hand in writing; in some cases of writer's cramp the disturbance consists in a similar to-and-fro tremor of the distal segment of the limb which is accompanied by absolute stationariness of the pencil-point. In various other occupation neuroses tremor or tremulousness manifests itself.

Mention should be made of certain other phenomena not infrequently seen. There may be a painful and distressing sensation of "dragging," "drawing," "tightening" in the muscles involved, of which the patient is aware but which does not exteriorize itself. Tingling, numbness, and other paræsthesiæ are sometimes complained of. The paræsthesiæ may amount to actual pain, which in some cases seems to radiate from the site of the affected muscles and extend into other segments of the limb. Often the chief subjective sensory symptom is a dull aching of the whole limb, a constant feeling of tiredness and of muscle fatigue, which in severe or aggravated cases is noticed also when the muscles are being used for other purposes. In certain of these cases, however, there may be some more definitely organic basis for the symptoms.

Pathogenesis.—The fact that usually the patient is able to employ the affected muscles for any other purpose than that which constitutes the "occupation" seems to show

that the seat of the trouble is not peripheral or medullary, but cerebral. The preliminary to the ordered execution of any complex of fine movements is the education and co-ordinated innervation of a corresponding series of inter-related nerve-centres. In predisposed individuals, persistent repetition of the movements when fatigue has set in, early it may be, leads to exhaustion of the nerve-centres. Coupled with this exhaustion is a sense of fatigue; the sensory stimuli accompanying the movement become unduly prominent, occasioning discomfort; finally, as Starr says, the mechanism revolts.

It is no doubt conceivable that an exhaustion neurosis may have its basis in, or lead to, some molecular structural change in grouped nerve-cells; at the same time, it is difficult to conceive how, immediately after failure of innervation, as in writing, identically the same muscles may be innervated, say, for piano playing. Recognizable pathological changes in cases of occupation neurosis have not hitherto been found, and it is questionable whether they ever will.

Diagnosis.—The diagnosis should present little or no difficulty. It is important to realize that an occupation neurosis, occurring with neurasthenic symptoms, is nevertheless to be regarded as a separate affection, unless it can be shown to be merely a consequence of neurasthenic tremulousness. Occasionally *agraphia* is wrongly diagnosed as writer's cramp. Similarly, the latter condition may be diagnosed when the condition is really one of *motor apraxia*. Various organic nervous diseases, moreover, such as *paralysis agitans*, *tobes*, *disseminated sclerosis*, may in their earliest stage result in disturbances of co-ordination, evidenced in attempting to write, which may mislead the practitioner into imagining that he is dealing with writer's cramp.

Prognosis.—The prognosis, unfortunately, cannot be regarded as favourable, in spite of the comparative innocuousness of the condition. Some cases make a good recovery, others remain more or less intractable; in all alike relapses are frequent.

Treatment.—In *writer's cramp* the prime essential is rest, it may be for many months, from the movements concerned, though the patient may be allowed to use the affected muscles for any other purpose. At the same time he should learn to write with the left hand. Cessation for six months or so may in some instances bring about a cure, and the condition

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may never return. When writing is resumed the patient should begin more or less after the fashion of children, writing "big, round, and slow." If the case is mild in degree, or if writing of a sort is still practicable, he may obtain help from some simple apparatus. He should cultivate a freer style of writing, making the movements more from the shoulder and less from the fingers. He may use a thick cork penholder, so as to lessen the strain on closely approximated fingers. A pencil or a stylographic pen is better than a steel nib. Sometimes the mere holding of the pen between the first and second fingers is of service. More elaborate devices are Nussbaum's bracelet, and a ring attached to the pen, which may be passed over the index finger.

Drugs are of little or no use. Local treatment to the forearm and hand is in many cases of distinct value. Galvanic baths or galvanofaradism can be recommended. In addition, massage and exercises for the groups involved should be persevered with. Whether such forms of treatment are merely suggestive, or actually exercise a local effect, is immaterial. There is no doubt that the average type of patient is impressed by treatment which deals with what he imagines to be the seat of affection, and in no case should such treatment be omitted for merely pedantic reasons. Hydrotherapy and other general measures should be adopted if the patient's neurasthenic state calls for them.

The treatment of other varieties of occupation neurosis is not essentially different. Should the bowing hand be implicated in violinist's cramp, the stem held in the fingers may be widened by gluing on a suitable piece of cork so as to lessen the degree of continual tonic contraction of the small hand-muscles. In telegraphist's cramp the instrument is usually so placed that the operator's arm is unsupported; if this can be placed on the table the strain is often materially reduced. In pianist's and other cramps that might be mentioned it will be clear to the practitioner that no simple preventive device can be invented; in such cases the sole treatment is insistence on absolute rest, coupled with the general therapeutic measures indicated above.

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follow an injury may be perpetuated by auto-suggestion as hysterical symptoms after the organic changes have more or less completely disappeared. The emotion of fear, which often results from the events that gave rise to the injury, produces physical manifestations, which may also be perpetuated by auto-suggestion as hysterical symptoms. The incapacity resulting from the accident often leads to considerable financial worry, which is intimately associated with the question of compensation. At the same time the forced inactivity and the pain which may be present exert a depressing action on the nervous system. The symptoms which result from these conditions are generally described as traumatic neurasthenia, but they are really a combination in varying proportions of neurasthenia, hysteria, and psychasthenia, the second and third being generally the most important. They are chiefly mental in origin, and disappear when the worries are no longer present, after recovery from the physical disability and the settlement of claims for compensation.

The whole question is complicated by the frequency of malingering (q.v.). Though pure malingering is rare, a man often exaggerates the severity of his pain and the degree of his incapacity; he refuses to acknowledge improvement when improvement is taking place, and does not co-operate with the physician in his efforts to cure him.

Symptoms. The symptoms of concussion which follow a blow on the head or spine rapidly disappear with rest, unless the injury is severe. If the headache caused by concussion of the brain persists, this is due either to insufficient rest in the early stages or to the concussion headache being perpetuated as an hysterical headache owing to auto-suggestion, to which is often added the hetero-suggestion of over-sympathetic friends. There is nothing characteristic about it, except that it rarely interferes with the patient's doing anything he really wishes to do, although it makes him quite incapable of working. In rare cases hemiplegia, hemianopia, and convulsions, which resulted from the organic changes in the brain caused by the concussion, may continue as hysterical symptoms after all evidence of organic injury has disappeared. Much more frequently the paraplegia, which results from the temporary changes produced by concussion in the spinal cord, is perpetuated in this way. Both the hemiplegia and the paraplegia have the same clinical

NEUROSES, TRAUMATIC.—Functional nervous disorders which result from physical injuries.

Etiology and pathogenesis.—The symptoms caused by the organic changes which

features as the organic condition which they follow, and which suggested the continued existence of the paraplegia. In most cases a diagnosis is easily made by the complete absence of physical signs of organic disease, but in some instances these may still be present owing to the persistence of a small organic lesion which is insufficient to produce any symptoms. The diagnosis can then only be made by seeing what improvement results from vigorous psychotherapy. In such cases, when the hysterical paralysis has been cured the organic physical signs of course remain.

The pain and the protective spasm of the muscles of the back which follow an injury very frequently persist in the same way as the "railway spine" of Erichsen and the "bent back of soldiers" which was so often met with in the War. Though due at first to bruising, they were eventually always hysterical, the particular position assumed being that which the patient originally found most comfortable. The stiff joints, contractures and paralyses of the hand and foot following comparatively trivial injuries of the limbs are often purely hysterical, though disuse may lead to such deficient circulation that secondary trophic changes may occur in the skin, bone, and nails.

The hysterical tremor, mutism or stammering, and paraplegia which are caused by the perpetuation of the physical manifestations of fear are rare in civil life, though they are occasionally observed after railway accidents, but they were common sequelæ of the much more terrifying events of war.

The chief psychasthenic symptoms which follow accidents are depression and phobias. The problems the patient is unwilling or unable to face during the day assume a more overwhelming aspect at night, and lead to insomnia and nightmares, which in turn are followed by headaches and all the other symptoms of neurasthenia.

Treatment.—A thorough examination must be made in order to determine the extent of the actual injury, and at the same time to inspire the patient with confidence that his case is being taken seriously and judged on its merits apart from any question of compensation. A firm but sympathetic attitude, with a minimum of physical and medicinal treatment, should thus, in the large majority of cases, prevent the development of the traumatic neuroses. When they have once developed they should be treated by explanation and

encouragement, persuasion and re-education. Suggestion should be avoided, whether indirect by means of drugs or electricity, or direct with the aid of hypnosis, as it assumes that there is some more or less serious condition present which requires this treatment. On the other hand, the responsible authorities should be made to understand that though the patient's troubles are not organic they are very real, and that the best way to cure him rapidly and permanently is to compensate him generously at once with a lump sum, and not to pay weekly doles, which do not encourage him to get well, and which are likely to lead to litigation, and through this to the aggravation of his psychasthenic symptoms.

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NEUROSES, WAR.—The two conditions which led during the War to the great frequency of neuroses in soldiers, compared with their comparative rarity in men under peace conditions, were exhaustion and emotional strain. The exhaustion caused by long days of forced marching or strenuous fighting, followed by nights with little or no sleep, combined in some cases with insufficient food, and in the Eastern campaigns with a great variety of infections and exposure to extreme heat, naturally led to a more profound neurasthenia than is commonly seen in civil life. In spite of this, the intervals of rest and opportunities of relaxation, which became increasingly common as the War progressed, together with the admirable supply of food in most cases and the freedom from epidemic infections on a large scale owing to the excellence of the sanitary arrangements—except at Gallipoli and in the earlier part of the Mesopotamian campaign—prevented it from being as widespread or as severe as might have been expected. More important, perhaps, than the actual production of neurasthenia as a result of exhaustion were the increased liability to the development of other neuroses, such as hysteria and psychasthenia, and the aggravation of incipient organic diseases, such as general paralysis and tabes.

Whilst it is true that a few fortunate individuals are born with a temperament which does not allow them to know what fear means, the vast majority of men, including many of the bravest, are terrified when first exposed to the horrors of a bombardment. In time the majority became accustomed to it, but the exhaustion of active service often resulted, sooner or later, in a gradual failure of the

NEUROSES, WAR

adaptation, so that not only the constitutionally timid—the “martial misfits”—but some of those who for months or even years had faced the life cheerfully, and even with enjoyment, ultimately broke down from long-continued emotional strain.

The emotion of fear acts in three ways. In the martial misfit, who is by nature very suggestible, it gives rise at once to severe physical symptoms which often become perpetuated by auto-suggestion, such as hysterical tremor, mutism or stammering, paraplegia, and fits; in other cases it gradually leads to the development of psychasthenia; and finally it may result in such a disturbance of the sympathetic nervous system that severe symptoms involving the cardio-vascular, digestive, or genito-urinary systems are produced, which may be associated with excessive secretion of the thyroid and suprarenal glands.

Apart from the fear which is caused by the general conditions of a prolonged bombardment, the more acute emotion excited by a single exceptionally terrifying experience leads to such a disturbance of mental equilibrium that the subject becomes for a time extremely liable to develop hysterical symptoms by suggestion. This is particularly true if the experience has had actual physical results; however evanescent they may be, they are likely to be unconsciously perpetuated and even exaggerated by the patient. Thus, a man who is gassed may develop hysterical blepharospasm, ptosis, and blindness after the initial conjunctivitis has disappeared, hysterical aphonia after the disappearance of the laryngitis, or hysterical vomiting after the disappearance of the gastritis. If a man is blown up or buried, the amnesia, headache, hemiplegia, or convulsions which may result from concussion of the brain, the deafness from concussion of the internal ear, and the paraplegia and incontinence of urine from concussion of the spinal cord, may be perpetuated as hysterical symptoms after the actual changes in the nervous system have so greatly diminished in degree and extent that the symptoms should have completely or almost completely disappeared.

Prognosis.—The prognosis of the war neuroses was excellent if the treatment was initiated at a very early stage in advance hospitals, and numerous patients were able to return to the front line within a few days and carry on without fear of relapse. The prognosis became progressively worse after the patient reached a base hospital abroad, a general

hospital in England, and, worst of all, V.A.D. hospitals, where the sentimental sympathy and absence of discipline had the worst possible effect. But, even in cases which had been mismanaged for months or years, recovery was comparatively rapid in the special neurological hospitals which were finally established in England. This was especially true of hysteria, cases of which could often be cured completely in a single sitting, however long the symptoms had persisted. The prognosis of neurasthenia also was good, but the psychasthenic soldier, when he had once reached England, rarely became fit for active service again, though with proper treatment for a sufficient period he would probably always have recovered, if suitable work could at once have been found for him after his discharge from the service.

Treatment.—This does not differ essentially from the treatment of similar conditions seen in civil life. A very short period of rest, followed by graduated and interesting work, with psychotherapy in the form of explanation, persuasion, and re-education, but including a minimum of suggestion, whether in the waking state or under hypnosis, was all that was required. Drugs were hardly ever needed, except a little bromide for excitable patients, and gradually reduced doses of hypnotics during the first days of treatment in severe cases of insomnia. The most successful medical officers were those who were able to gain their patients' confidence by their firm but sympathetic treatment, and at the same time imposed the right amount of discipline, without which the most scientific psychotherapy was likely to fail.

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NEW GROWTHS (see under individual organs).

NIGHT BLINDNESS (see DAY BLINDNESS AND NIGHT BLINDNESS).

NIGHT TERRORS (see SLEEP, DISTURBANCES OF).

NIPPLE, AFFECTIONS OF (see BREAST, AFFECTIONS OF).

NOCTURNAL EMISSIONS (see SEXUAL FUNCTIONS, MALE, DISTURBANCES OF).

NOCTURNAL ENURESIS (see INCONTINENCE OF URINE).

NODAL RHYTHM (see HEART-BEAT, ABNORMALITIES OF).

NOSE, EXAMINATION OF

NODDING SPASM (*see* SPASMUS NUTANS).

NOMA ORIS (*see* STOMATITIS AND GLOSSITIS).

NOMA VULVÆ (*see* VULVA, DISEASES OF).

NOSE, ACCESSORY SINUSES OF (*see* SINUSES, ACCESSORY AIR, DISEASES OF).

NOSE, BLEEDING FROM (*see* EPISTAXIS).

NOSE, EXAMINATION OF.—The patient should be seated on a chair or stool facing the examiner in a darkened room. The lamp should be placed on his left side and on the same level as his head. The forehead mirror should have a focal distance of about 12 in. and a large oval or round hole in its centre; this hole should not be less than half an inch in diameter, as a smaller aperture leads to difficulties in examination.

1. The first step consists in the **examination of the exterior of the nose**, such conditions as deformities, eczema, or lupus being noted.

2. **Anterior rhinoscopy** by means of a Thudichum's speculum should then be proceeded with. The patient's head must be in such a position that the examiner is able to look straight back along the floor of the nose.

The examiner's hand should rest lightly on the patient's nose, for unless this be done, any movement of the patient's head is likely to cause the speculum to slip out of position, so that the examination has to be begun again.

The interior of the *vestibule* is first inspected to detect such lesions as growths, ulcers, furuncles, etc.

Next, the *nasal septum* is examined for the presence of pathological conditions or of deformities. Normally it is vertical and covered with pinkish mucous membrane.

If the septum is deflected it will be seen to project into the lumen of the nostril, or if the concave side of the deflection is under examination it will be shown by a curving away of the septum from the middle line into the opposite nostril.

A spur can easily be distinguished from a deflection by the absence of a corresponding concavity on the other side.

The *inferior turbinal bones* should now be examined. They also should be covered with pinkish mucous membrane, and there should be a clear airway of about $\frac{1}{4}$ in. between the septum and the inferior turbinal bone, and again between the inferior turbinal bone and the floor of the nose.

The presence of pus or growths, and atrophic or hypertrophic states, should be noted.

The examiner should then turn his attention to the *middle turbinal bone*, and for this purpose the patient's head should be tilted slightly backwards. There should be a clear airway between the middle turbinal bone and the septum, and between it and the inferior turbinal bone.

Obliteration of the space between the middle turbinal bone and the septum results in loss of smell, and any pressure in this region may also be responsible for intense headaches or asthmatic symptoms.

Pus passing down over or under the middle turbinal bone must be carefully looked for. In the former position it denotes suppuration of the sphenoidal sinus or posterior ethmoidal cells; in the latter, suppuration in the frontal sinus, in the anterior ethmoidal cells, or in the maxillary antrum.

3. **Examination of the postnasal space.**

(a) **Posterior rhinoscopy.**—The patient's mouth should be opened widely and the tongue depressed by a Fränkel's or Lack's depressor. Firm, steady pressure must be maintained, for it is the repeated movement of this instrument over the tongue which produces retching or coughing and makes examination impossible.

Some people are quite unable to tolerate a tongue-depressor or postnasal mirror until the palate and the posterior wall of the pharynx have been sprayed with a 5-per-cent. solution of cocaine.

The patient should next be instructed to breathe quietly through the nose, otherwise the soft palate will not fall forward, nor will the postnasal area come into view. The mirror, which has been previously warmed over a spirit lamp, should be inserted well down into the pharynx in such a way that the practitioner is able to see the structures in the postnasal space.

Accidents often happen, and the patient's confidence is lost, if the mirror is inserted when it is too hot; to obviate this the back of the mirror should be previously applied to the examiner's forearm.

Should the patient be a child, the warming of the mirror must always be done where it cannot be seen, otherwise it will be assumed that some burning operation is contemplated; no amount of persuasion will then overcome this belief, and the examination may thus be rendered impossible.

The mirror must not be allowed to come into

NOSE, EXAMINATION OF

contact with the base of the tongue or the posterior wall of the pharynx, lest retching be provoked and lead to failure in obtaining a view of the naso-pharynx.

A long examination should be avoided, as it is very trying for the patient; after a short time the mirror should be withdrawn, and, if the examination has not been completed, the instruments can again be inserted after the patient has had a few minutes' rest.

The first structure to be identified is the posterior border of the soft palate, which appears on the lowest part of the mirror. The posterior border of the septum is seen running upwards from its central point. Just above the palate and on the outer side of the posterior choanae the posterior ends of the inferior turbinal bones are seen, and above these the posterior ends of the middle turbinal. Normally there is an airway between the posterior end of the inferior turbinal bone and the septum, and between the same bone and the floor of the nose. It may be possible to identify the orifices of the Eustachian tubes.

The structures having been identified, any deviation from the normal, such as the presence of polypi, growths or pus, should be noted.

Adenoids appear in the upper part of the mirror as an irregular mass varying in size according to the amount of growth; they prevent the upper part of the posterior border of the septum from being seen.

(b) **Digital examination** is only called for in children when the postnasal mirror cannot be used; owing to its painful nature and the terror which it produces it should only be used as a last resort.

When deflections of the septum are present, when the inferior or middle turbinal bones are enlarged to a sufficient extent to require operation, or when enlarged tonsils obviously requiring removal are present, the postnasal space can be examined digitally under an anæsthetic, and any adenoid mass or enlargement of the posterior ends of the inferior turbinal bones dealt with.

If the child has no obvious nasal or pharyngeal condition which demands operative treatment, and is of very nervous temperament, examination under an anæsthetic is preferable; any defect can then be dealt with surgically without delay.

When it is found necessary to resort to this form of examination without an anæsthetic, the following method should be employed: The patient should be wrapped in a blanket

NOSE, MALIGNANT GROWTHS OF

to prevent struggling, and seated on the parent's or nurse's knee. The practitioner places his left arm round the child's neck, and tells him to open his mouth as widely as possible. He is then able, with his left hand, to push the cheek between the upper and lower teeth and at the same time to hold the child's head firmly against his side. Next, the first finger of the right hand is passed quickly into the naso-pharynx and the examination carried out, care being taken not to remove the left hand from the cheek until the examining finger is withdrawn, otherwise the examiner is almost certain to be badly bitten.

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NOSE, FOREIGN BODIES IN (see FOREIGN BODIES IN THE AIR-PASSAGES).

NOSE, INFLAMMATION OF MUCOUS MEMBRANE OF (see RHINITIS).

NOSE, LARVÆ IN (see MYIASIS).

NOSE, MALIGNANT GROWTHS OF.

The forms most commonly met with are sarcoma, epithelioma, and alveolar carcinoma. At the onset, slight nasal obstruction, neuralgic pains, and attacks of epistaxis are usually complained of. In the later stages the pain increases, the obstruction becomes much greater, and a foul discharge makes its appearance. The bleeding becomes more frequent and may be profuse. As the growth extends and invades the surrounding structures, external deformity, ocular symptoms due to invasion of the orbit, and intracranial symptoms due to invasion of the cranial cavity may all be present. Examination of the interior of the nose reveals a fungating, irregular mass, which may be ulcerated and may have perforated the septum; it always bleeds freely on being touched with the probe.

Diagnosis.—Malignant nasal growths must be diagnosed from innocent growths and from tuberculous, lupoid, and syphilitic lesions. In advanced cases the appearance of the growth and the way in which it has invaded the surrounding structures are sufficiently characteristic. A certain diagnosis, however, can only be made by the removal of a portion for microscopic examination; this should be done in all cases.

Treatment.—Unfortunately, the patients do not usually seek advice until the disease is fairly advanced, so that many cases have progressed too far for a complete removal to

be undertaken; in these, palliative treatment is alone possible. When the disease is in an early stage, or when, though more advanced, there is a possibility that the growth can be removed, operative treatment with a view to complete eradication should be undertaken at once.

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NOSE, SYPHILIS OF.—Nasal lesions are met with both in acquired and in congenital syphilis.

Acquired syphilis.—A *primary sore* is very rarely seen in this situation; its presence gives rise to slight obstruction, pain in the affected nostril, and occasionally a slight discharge. In the *secondary stage* mucous patches are seen very occasionally; the mucous membrane in this phase of the disease may be somewhat injected and inflamed. In the *tertiary stage* gummata may develop, their commonest site being the septum; they ulcerate very quickly, and the septum is rapidly destroyed. Obstruction progresses quickly, and when ulceration takes place is accompanied by a foul discharge and by attacks of bleeding. Headache and pain in the nose are complained of. When the disease is advanced, crusts appear, and pieces of dead bone are detectable. With invasion of the nasal bones the bridge of the nose becomes flattened and deformed. Perforations of the hard and soft palate may also appear.

Congenital syphilis.—The commonest form occurs soon after birth, and is indicated by profuse purulent discharge. Ulceration occurs sooner or later, and by spreading to the nasal bones may cause great deformity ("saddle-back" nose). In advanced cases the septum and turbinal bones are often almost completely destroyed.

Diagnosis.—In all forms of the disease a positive Wassermann reaction, combined in acquired cases with signs of the disease elsewhere, and in the congenital variety with the presence of condylomata in the usual positions, makes the diagnosis easy. Any doubt will be removed by the effect of specific treatment.

Treatment.—In addition to the usual general measures, local treatment may be useful. If there is much crust-formation, the nose should be irrigated two or three times a day with saline solution, followed by spraying with an oily solution such as—

R̄ Menthol gr. v.
Ol. eucalypt. ʒiii.
Ol. cinnam. ʒv.
Paroline ad ʒi.

Pieces of dead bone should be removed with forceps as soon as they are completely separated.

When the disease has become arrested, the turbinal bones can sometimes be built up again by injections of solid paraffin sub-mucously. If this can be done, much of the discomfort due to crust-formation can be alleviated. In most cases, however, an alkaline douche and an oily spray will have to be used permanently.

G. N. BIGGS.

NOSE, TUBERCULOSIS OF.—Although occasionally primary, nasal tuberculosis in most cases is secondary to tuberculous lesions in other parts of the body, especially in the lungs. The symptoms usually complained of are nasal obstruction, attacks of bleeding from the nostrils, a muco-purulent or purulent discharge, and the formation of crusts in the nostrils. The accessory nasal sinuses are often invaded in the later stages of the disease.

Several forms are met with. One or more shallow ulcers may be seen on the mucous membrane in any position, but usually on the septum. In other cases a red swelling may be present. The commonest lesion is a perforation of the septum. In some places the edges of the perforation may show signs of active disease, as evidenced by the presence of granulation tissue, whilst in others the disease has healed; this appearance is important in diagnosis, for it is rarely seen in other varieties of perforation.

Diagnosis.—The disease has to be distinguished from syphilis, lupus, innocent and malignant growths, and leprosy. A piece of diseased area or fragments of granulation tissue should be removed for a microscopical examination; this will usually make an accurate diagnosis possible. The lesions most likely to cause difficulty are those of *syphilis*; usually, however, syphilitic perforations involve the bony septum as well as the cartilaginous portion, whereas those caused by tuberculosis are more often confined to the latter. In syphilis, again, the disease is much more rapid, there are signs of syphilis elsewhere, and the local condition quickly reacts to antisyphilitic treatment. *Lupus*, as a rule, can easily be distinguished by the presence of the nodules in the skin and by the characteristic scarring and deformity of the nostrils. The granulations in lupus are smaller and paler, and the lesions progress very slowly, typical scarring being present in some parts of the perforation and

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active disease in others. If a weak solution of cocaine be applied to the mucous membrane, lupoid nodules are recognized by their failure to change in colour, and can be seen as red areas on the whitish mucous membrane. In advanced *leprosy* there may be extensive destruction of the septum, as in nasal tuberculosis; but in the former disease nodules will usually be found somewhere on the nasal mucous membrane, and sections of these will demonstrate the presence of the leprosy bacillus. The presence of leprosy lesions elsewhere renders the diagnosis easy in most cases.

Treatment.—Complete excision should always be carried out when possible, and afterwards the edges of the wound should be painted daily with the following pigment:—

R̄	Acid. lact. (B.P.)	partes 40.
	Formalin (B.P.)	„ 7.
	Acid. carbol.	„ 8.
	Aq. ad	„ 100.

Crust-formation can be reduced to a minimum after the wounds have healed by douching the nose frequently with a saline solution, followed by spraying with such oily solutions as menthol in paroline 5 per cent., or—

R̄	Menthol gr. v.
	Ol. eucalypt. ʒiij.
	Ol. cinnam. ʒv.
	Paroline ad ʒi.

Patients must attend regularly for examination in order that recurrences may immediately be dealt with.

If operation cannot be carried out or is refused, as much as possible of the lesion should be destroyed with the cautery and the pigment mentioned above applied daily.

Prognosis.—When the diseased parts can be removed completely the outlook is good, provided that the condition is a primary one; when it is secondary to tuberculous disease elsewhere, the prognosis will depend on the severity and extent of the primary lesion.

G. N. BIGGS.

NOTIFICATION OF INFECTIOUS DISEASES.

By the Infectious Diseases Notification Act of 1889, every practitioner attending a case of smallpox, cholera, plague, diphtheria, membranous croup, erysipelas, scarlet fever, typhoid (enteric) fever, typhus, relapsing fever, or puerperal fever must notify the same to the Medical Officer of Health for the district. Cerebro-spinal fever (epidemic cerebro-spinal meningitis), in which is included pos-

NYSTAGMUS

terior basic meningitis, and acute poliomyelitis and polioencephalitis were added to this list in 1912; in 1914 the further addition of ophthalmia neonatorum was made; and in 1919 malaria, dysentery, trench fever, encephalitis lethargica, acute primary pneumonia, and acute influenzal pneumonia were also made notifiable. The notification of tuberculosis is dealt with in TUBERCULOSIS; A GENERAL SURVEY FROM THE STANDPOINT OF PREVENTIVE MEDICINE.

The Sanitary Authority, with the sanction of the Local Government Board, has the power to make other diseases notifiable, either permanently or for a specified time only. During an epidemic of smallpox, varicella is often made notifiable, as a mistaken diagnosis of chickenpox has more than once been responsible for the dissemination of the more serious disease. Measles and whooping-cough, which are not generally notifiable, have been made so in certain districts owing to their high mortality in young children.

Certificate forms are issued free by the local authorities. A fee of 2s. 6d. is allowed for cases in private practice, and a fee of 1s. for cases in public institutions. A penalty of 40s. may be inflicted in case of neglect to notify, to which the householder is also liable if, in the absence of a doctor, having reason to believe that a person in his house is suffering from one of the above-named infectious diseases, he fails to notify it.

J. D. ROLLESTON.

NUTRIENT ENEMATA (see ENEMATA).

NYCTALOPIA (see DAY BLINDNESS AND NIGHT BLINDNESS).

NYSTAGMUS.—This term is applied to various types of involuntary oscillations of the eyeballs, which may be present in any or every position of the eyes, or may appear only when they are deviated in one or other direction.

Two main forms are distinguished: the undulatory or pendular type in which the excursions are equal to each side of a central point, and the rhythmical in which there are two phases, a slow deviation from the point fixed and a quick jerk of the eyes in the opposite direction. The slow deviation is the essential feature, but the direction of the nystagmus is generally described as that of the quick phase. Both eyes generally move synchronously and equally, but occasionally nystagmus is unilateral. In a rare form it appears in one eye only when the other is closed.

Nystagmic oscillations are most common in the horizontal plane, but they are frequently rotatory, or may be vertical or irregular. Occasionally, but particularly in early life, they are associated with tremulous or jerky movements of the head which may be compensatory. As a rule the patient is not conscious of apparent movement of objects which he sees, except in recently acquired or severe nystagmus, as in that of labyrinthine origin. It is then associated with vertigo.

There are many causes of nystagmus, but they may be grouped into ocular, labyrinthine and nervous. Miner's nystagmus is considered separately (*see* MINER'S NYSTAGMUS).

Ocular nystagmus is usually of the undulatory type. The congenital is the most common form. It is the result of amblyopia due to cataract, leucomata, macular lesions, high myopia, or other causes that prevent the formation of clear images on the infantile macula. Ocular nystagmus is also common in albinos, in whom the retinal pigment is defective; the hereditary form is probably due to this. It occurs in spasmus nutans too; the oscillations of the eyes usually appear later than the head movements, in about the third month, and frequently disappear during the third year.

Labyrinthine nystagmus is produced by stimulation from rotation, galvanization, and sudden alterations of temperature and pressure in one labyrinth, or it may be due to destruction of the internal ear on one side. It is of the rhythmical type, and is generally rotatory.

When the lesion is destructive it consists in a slow deviation of the eyes to the affected side and rapid jerks in the opposite direction. It is most marked on looking to the contralateral side. It is usually associated with vertigo.

Nystagmus of nervous origin is most commonly due to lesions of the central connexions of the vestibular nerve, or of the cerebellum or its peduncles. It is consequently found chiefly with disease in the posterior fossa of the skull. The main features of nystagmus due to a unilateral cerebellar lesion are: It is rhythmical, but as a rule occurs only on lateral deviation of the eyes; the oscillations are slower and coarser on deviation in the homolateral than in the opposite direction, and it consists of a slow phase that tends to bring the eyes towards the primary central position, and rapid jerks in the direction towards which they are voluntarily deviated.

Nystagmus also occurs in many nervous diseases, as disseminated sclerosis, Friedreich's ataxia, and syringobulbia.

Nystagmic jerkings of the eyes may occur in normal persons when they strain to maintain the visual axes in extreme lateral deviation beyond the binocular field. It may be merely a hesitancy in fixation. This type is common when there is any paresis or asthenia of the ocular muscles, and in alcoholism and acute fevers.

GORDON HOLMES.

NYSTAGMUS, MINER'S (*see* MINER'S NYSTAGMUS).

OBESITY. — A disorder of metabolism characterized by excessive deposit of fat in the body (Osler).

Etiology and pathology.—It is not easy to say exactly when a deposit of fat passes the physiological limit, but it is certain that what constitutes pathological obesity for one individual does not for another. Stoutness which does not interfere with health or efficiency is not a disease. Some races tend to become fat, and a similar tendency is seen in families.

In one group of cases obesity is a sign of *defective sexual development*. Castration gener-

ally causes obesity, and the involution of the reproductive apparatus at the menopause is frequently accompanied by an increase of fat. The extraordinary obesity sometimes seen in boys at puberty is usually a sign of delayed sexual development, and may subside completely a few years later. Recent work suggests that in such cases there is a defect in the internal secretion of the pituitary gland, which is known to enlarge during pregnancy. Fröhlich's syndrome of hypopituitarism (obesity, increased carbohydrate tolerance, and hypoplasia of the reproductive organs) is a similar

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but more extreme condition, dependent on an organic lesion of the gland. The obesity is probably the result of the increased tolerance of carbohydrates, which, in the absence of the more active metabolism induced by the internal secretions of the reproductive organs, are not consumed but deposited as fat. (*See PITUITARY GLAND, AFFECTIONS OF.*) In pituitary obesity there may be uncontrollable somnolence—as in the fat boy in “Pickwick.” There is also antagonism between the thymus and the reproductive organs, and the subjects of status lymphaticus are usually plump. In thyroid inadequacy, too, fat may be increased, but probably only proportionately to the increase of other subcutaneous tissues. The type of obesity here described may really be due to polyglandular insufficiency, but, for the present, sexual hypoplasia is the outstanding feature.

Usually the deposit of fat due to defective secretion is general, but in adiposis dolorosa (Dercum's disease), which is also thought to be due to some such defect, the deposit is local and symmetrical on the limbs and trunk, though not on the hands, feet, or face. (*See ADIPOSIS DOLOROSA.*)

Apart from these somewhat rare conditions it can hardly be doubted that ordinary obesity of middle life is due to a disproportion between the intake and output of energy. It need not be due to excessive intake, for many stout people are quite moderate eaters. In that case they are generally not physically active, but placid in mind, contented in disposition, and lethargic in movement. There is a tendency to early emphysema, causing defective oxidation and further disinclination to exertion. Even here diet may be concerned qualitatively rather than quantitatively, for such persons may display a preference for sweet, starchy, and fatty foods. The rapid development of obesity sometimes seen after typhoid fever is largely due to enforced rest during a tedious convalescence, but may be in part due to toxic action on the thyroid and pituitary glands.

With the slowing of metabolism which occurs after 40, we probably all tend to eat too much. The average allowance of Calories for the day's work is 2,800; if the food of the day contains more than this, or if the physical work of the day consumes less, there must be a balance which is deposited as fat.

Alcohol plays an important part in producing obesity; it provides Calories and, being an oxidizable substance which is very diffusible,

obtains the first call on the oxygen, so that the oxidation of the other foodstuffs tends to be less complete. Moreover, malt liquors contain carbohydrate, and are taken irrespectively of the occurrence of hunger.

Symptoms.—The deposit of fat entails the carrying about of an excess of inert material. This disposes to less exertion, and a vicious circle is thus established. Some of the fat is deposited on the heart, embarrassing its action and causing shortness of breath. This aggravates the vicious circle and makes treatment by physical exercise more difficult and sometimes even dangerous. In some persons obesity favours the development of raised blood-pressure and arterial degeneration. They are examples of the “bull-necked” variety, liable to cerebral hæmorrhage. In this type there may also be localized fatty deposits, or diffuse lipomata, forming a huge collar round the neck, especially in men who are beer-drinkers.

During rest the sweat-glands of the obese are less active than normal (von Noorden), but on comparatively slight exertion sweating may be excessive. The association of glycosuria with obesity is well recognized, yet according to von Noorden the glycosuria is not the result of obesity, but both are evidence of disturbed carbohydrate metabolism. At first the diminished capacity for consuming carbohydrate is met by converting it into fat, but, as there must be a limit to this process, an overflow of sugar into the urine ultimately occurs. In other words, in this type of obesity there is latent glycosuria.

Treatment.—As long as obesity is compatible with good general health, its reduction is not really called for, especially if reduction in weight is accompanied not by increased well-being but by the reverse. In any case, rapid and drastic reduction is seldom, if ever, advisable, for by it the patient becomes ill and miserable, and is unlikely to persist in treatment which he considers to be worse than the disease. Moreover, rapid reduction is by no means free from danger. It is sufficient if a reduction of 1 lb. a week for the first month, and subsequently of 1 lb. a month is effected. The ordinary diet should be inquired into, and, if it appear excessive in any particular, the necessary restrictions enforced. If the actual amounts be determined, the excess of Calories can be calculated and reduced to not more than 2,800. Since every lump of sugar means 20 Calories, the importance of giving up sweetened drinks can readily be appreciated.

OBESITY

Adherence to a few simple rules is usually sufficient. Sugar, honey, jams, pastry should be forbidden. The total amount of bread should be reduced to 2 oz.; if it be toasted it will go further. Kalari biscuits, such as are given to diabetics, are an excellent substitute for bread. With a little patum spread on them they are quite palatable at breakfast or tea. Potatoes, carrots, artichokes, and beet-root should not be eaten, but, on the other hand, green vegetables may be taken freely, especially the less assimilable forms, such as lettuce. If the stomach is filled with a good deal of cellulose in this way, the sense of repletion, to which it is accustomed, is still obtained, though the amount assimilated is reduced. A freer action of the bowels is also procured, but some discomfort may be caused by flatulence. No cream and very little butter should be allowed. It is usual to forbid drinking with meals, but probably the chief effect of this is to diminish the quantity eaten, as most people cannot eat dry food with relish. A glass of hot water on rising and on going to bed may be useful. The patient is better without alcohol altogether, but if he is unwilling to submit to this deprivation, the least injurious form is good whisky, well diluted and taken in strict moderation. Restrictions of this kind are generally sufficient to produce a gradual reduction of weight without risk or severe discomfort.

Increased output of energy should be aimed at, but it is worse than useless to start a stout person, with an epicardium loaded with fat, on violent and unaccustomed exercise. *Müller's respiratory and other exercises* should be carried out for a quarter of an hour every morning. As it is of the highest importance to strengthen the abdominal muscles, so as not to leave the viscera unsupported as the fat disappears, some of the exercises should be specially designed to this end. A simple plan is for the patient to lie flat on the floor with his feet under a solid piece of furniture, such as a chest of drawers; with his arms folded across the chest, and his legs straight, he should alternately raise his trunk to a right angle with his thighs and let himself down flat again. This exercise should be repeated six times, and, after a few days, ten times. Next, the hands should be clasped behind the neck and the lifting of the trunk repeated six times in this position. Finally, the exercise is repeated with the arms fully extended above the head. This provides a series of exercises of increasing

difficulty. In recent years *Bergonié's method* has attained considerable vogue. The patient is seated in a special chair and sand-bags are placed over groups of muscles. Electrical currents are then passed through the body so that the muscles are kept contracting against the resistance of the bags. Considerable work is thus done without much fatigue. Loss of weight can be quickly effected, but this will be regained on the cessation of treatment, unless dietetic restrictions are observed. Massage and electric-light baths may be helpful. Turkish baths are often useful, but should not be persisted in if cardiac distress is experienced in the hot room. Spa treatment is useful if the patient lacks self-control or opportunity to carry out treatment at home; for this purpose Harrogate, Bath, Vichy, Vittel, Evian, or Aix-les-Bains may be recommended.

Drug treatment really plays no part in the treatment of obesity, unless thyroid insufficiency exists, when thyroid extract is indicated. For cases with Fröhlich's syndrome I have given thyroid combined with pituitary extract with some success. In the treatment of ordinary obesity, thyroid extract is inadvisable. Much of the loss of weight is achieved merely by dehydration of the fats, and is therefore temporary. Moreover, the extract accelerates without augmenting the heart-beat, and as the heart is often overloaded with fat, it may be incapable of standing this extra strain. Further, a latent glycosuria may be roused into activity by the drug. Most proprietary remedies for obesity are composed of citric acid or seaweed. There is no justification for their use.

Diet.—Special diets, such as Banting's, Salsbury's, Oertel's, and Ebstein's, can be found described in works on dietetics. They are not further referred to here because I do not believe such systems are physiologically sound. If for any special reason, and after due consideration of the risks, it is decided to try a plan of more rapid reduction than the one here suggested, von Noorden's system of frequent but small meals might be resorted to. His diet, which contains 1,366 Calories, is as follows:—

- 8 A.M. Cold lean meat, 3 oz.; bread, 1 oz.; tea or coffee with a spoonful of milk and no sugar.
- 10 A.M. One egg.
- 12 noon. A cupful of strong soup without fat.
- 1 P.M. A small plate of clear soup; lean meat or fish, 5 oz.; potato, 3½ oz.; green vegetables; fresh fruit, 3½ oz.

OBSESSIONS

- 3 P.M. A cup of black coffee.
4 P.M. Fresh fruit, 7 oz.
6 P.M. A glass of skimmed milk.
8 P.M. Cold lean meat, $4\frac{1}{2}$ oz., with pickles;
Graham bread, 1 oz.; two or three
spoonfuls of fruit cooked without sugar.

Von Noorden allows two glasses of wine daily with this; the objections to alcohol have, however, been explained. The advantage of his plan is that it checks the development of a robust appetite.

W. LANGDON BROWN.

OBSESSIONS.—An obsession is a mental process which obtrudes itself into consciousness with apparently irresistible force, and whose presence is distasteful to or actively resisted by consciousness as a whole. The mental process concerned may be of various kinds, and obsessions are therefore subdivided into obsessive thoughts, actions, impulses and fears. Thus, a patient may be impelled to picture constantly to himself some horrible event (obsessive thought), or to carry out repeatedly some absurd action, e.g. to pick up every match he sees, and to scrutinize with the utmost care every room and place in case a match might be there (obsessive action). Again, he may feel constantly impelled to commit suicide (obsessive impulse), or he may feel intensely afraid of being out alone (obsessive fear, agoraphobia). Obsessive thoughts, actions, and impulses are known by a great number of names—for example, impulsive thoughts, compulsive ideas, imperative ideas, irrepressible impulses, and so forth, while the obsessive fears are generally termed phobias. It must be clearly understood that all the various types of obsession are closely akin to one another, and that two or more generally occur together in one and the same case.

An essential feature of all obsessions is that the patient struggles against the obsessing process, and realizes perfectly that it is intrinsically absurd, irrelevant, or without basis in fact. These characters distinguish obsessions from other morbid mental phenomena with which they are sometimes liable to be confounded—e.g. delusions, impulsive action in dementia præcox and epilepsy. They are a characteristic feature of certain types of psychoneurosis, which are hence termed **obsessional or compulsion neuroses**. These types are included by Janet in his conception of “**psychasthenia**” (see **PSYCHASTHENIA**).

BERNARD HART.

CEDEMA

OBSTETRICAL PARALYSIS (see **SPINAL NERVES, LESIONS OF**).

OBSTRUCTION, INTESTINAL (see **INTESTINAL OBSTRUCTION; INTUSSUSCEPTION; VOLVULUS; INTESTINE, NEW GROWTHS OF**).

OBSTRUCTION, NASO - PHARYNGEAL (see **NASO-PHARYNGEAL OBSTRUCTION**).

OBSTRUCTION, ESOPHAGEAL (see **ESOPHAGUS, AFFECTIONS OF**).

OCCIPITAL NEURALGIA (see **NEURALGIA**).

OCCUPATION DISEASES (see **ANTHRAX; CAISSON DISEASE; GLANDERS; INDUSTRIAL MEDICINE; Lead Poisoning, under MULTIPLE NEURITIS; MINER'S NYSTAGMUS; PNEUMONOCOCCOSES**).

OCCUPATION NEUROSES (see **NEUROSES, OCCUPATION**).

OCHRONOSIS. A rare metabolic disorder causing blackening of the cartilages, fibrous tissue, and skin.

Etiology.—Ochronosis occurs in two groups of cases. In one it is an “inborn error of metabolism” and associated with alkaptonuria, a condition in which proteid metabolism is imperfect, homogentistic acid and uroleucic acid appearing in the urine, which blackens on exposure to light. This form is hereditary, congenital, and often familial. In the other it is accompanied by carboluria, and is due to the absorption of phenol and its derivatives. It has followed the application of carbolic acid to large ulcerated surfaces.

Symptoms.—Black patches appear in the skin, sclerotics, cartilage of the ears and about the knuckles. In thin patients the pigmentation may be seen in the tendons of the hands and feet. Apart from occasional arthritis and the disfigurement, there is no disability. Post mortem, a general blackening of cartilage, fibrous tissue and ligaments has been found.

FREDERICK LANGMEAD.

OCULAR PALSY (see **OPHTHALMOPLÉGIA**).

CEDEMA (*syn.* Anasarca; Dropsy).—Effusion of serous fluid into subcutaneous tissues. CEdema may be divided into general and local.

GENERAL CEDEMA

This form of edema is often associated with the transudation of fluid into the serous sacs,

causing hydrothorax, hydropericardium, and ascites.

Varieties.—1. **Cardiac oedema** results from right-heart failure and, to a great extent, is mechanically produced. It appears first, therefore, in the most dependent parts and those most remote from the heart. When the patient is walking about it is first seen above the ankles, and is often clearly limited below by the upper border of the boot. If the patient is in bed it may appear in the back or scrotum, but usually there is also oedema in distal parts of the extremities, especially the dorsa of the feet and hands. It is preceded by dyspnoea on exertion and other evidences of heart failure, and engorgement of the systemic veins is present. The signs of the cause of the heart failure may also be detected. It is important to remember that among these is renal disease, which may therefore produce a cardiac oedema independently or in combination with one which is characteristically renal in distribution.

2. **Renal oedema** is probably toxic in origin and, in contradistinction to cardiac oedema, is little affected by mechanical forces. It appears first in the looser subcutaneous areas such as around the eyes, especially below the lower lid, in the back forming the "lumbar cushion," and in the scrotum and about the penis. It may appear at the same time on the shins, but the early oedema of renal origin is not confined to the region of the ankles, as is so often the case in cardiac oedema. It is more pronounced and more frequent in acute Bright's disease and in chronic parenchymatous nephritis than in the chronic interstitial form, in which it is often confined to the eyelids. The oedema in chronic interstitial nephritis is more often cardiac than renal in kind.

3. **Hepatic oedema** is often limited to the legs, and is preceded by ascites. Dyspnoea follows but does not precede the abdominal effusion. Enlargement of the liver having the characteristic features of cirrhosis, or diminution of hepatic dullness, can be detected in most cases. This third classical form of dropsy was formerly regarded as due to portal obstruction but, for the most part, is probably toxic in origin. Ascites due to causes other than cirrhosis may produce a similar oedema of the legs by obstruction to the flow of blood in the inferior vena cava.

4. **Beriberi** causes an oedema which, when generalized, resembles that of Bright's disease. When limited it is especially prone to affect the

shins (see **BERIBERI**). An "epidemic dropsy" has been described in India and Mauritius; it resembles the dropsical form of beriberi, in which the neuritic symptoms are slight, but differs in being accompanied by fever and by an eruption, erythematous on the face and rubecular on the trunk and limbs. During the War groups of cases of oedema were recognized which by some have been identified with epidemic dropsy. Probably both the epidemic dropsy of Calcutta and Mauritius and war oedema are closely related to beriberi and, like it, result from deficiency of vitamins.

5. **Toxic oedema** is more common in childhood. It is generally accompanied by gastrointestinal disturbance, either diarrhoea or the passage of unhealthy and offensive stools. It resembles renal oedema, and there may be diminution in the quantity of urine passed, but no albumin is present. Very similar is the oedema which occurs in infancy, and is associated with the passage of urine of a very low specific gravity, though normal in quantity. The oedema often disappears when the specific gravity of the urine returns to normal. After scarlet fever general oedema without albuminuria has been described. It is doubtful whether such cases should be considered as examples of unexplained "toxic" oedema or of nephritis without albuminuria.

6. **Anæmia** is not infrequently accompanied by slight oedema about the ankles. When the anæmia is severe, as in pernicious anæmia, splenic anæmia, and leukæmia, the oedema may be considerable and attended by effusion into the pleural or peritoneal spaces.

7. Oedema may develop in **wasting diseases**, particularly in infants and children. In the former it is a common symptom of chronic diarrhoea and of the carbohydrate form of dyspepsia. In the latter it especially accompanies abdominal disease, such as tuberculous peritonitis and celiac disease. Tuberculosis is a common cause both in children and in adults, and carcinoma in adults. Always ominous, it is not invariably a precursor of death. It begins about the dorsa of the feet or hands, and spreads upwards along the limbs, to become generalized before death in some cases.

8. **Oedema neonatorum** appears in premature, feeble, or syphilitic infants, usually within the first forty-eight hours of life. The temperature is often subnormal, the pulse feeble, and the respiration so shallow and ineffectual that pulmonary atelectasis is present. The condition is generally fatal. Closely allied is **sclero-**

CEDEMA

œdema neonatorum, which also occurs in feeble infants within the first few days of life or, rarely, may date from birth. It is more common in cold climates, and is accompanied by a temperature as subnormal as 90-95° F., or even lower. It makes its appearance on the dorsum on the hands and feet, on the lower abdominal wall, and in the face, and may afterwards spread until the whole body is involved, except the front of the chest. The skin may be mottled, blue or waxy in appearance. The œdema usually pits by firm pressure, but sometimes the tension is so great that pitting is unobtainable. Death may occur within a few days, but not infrequently the course is more protracted and ends in recovery.

9. **Fœtal œdema** usually causes death of the fœtus, but the child may be born alive and survive for a few days. Its etiology is obscure; in some cases it is possibly explained by congenital malformation of the heart, vessels or blood, in others by increased blood-pressure within the placenta, for this organ is found to be large and œdematous. Renal disease in the mother is another possible cause, but no such association is constant.

Treatment.—The treatment of general œdema should be directed to its cause. Symptomatic treatment may, however, be called for in cardiac, renal, and hepatic cases, and may afford considerable relief. Rest in bed is necessary. The indications are to diminish the amount of liquids taken and to administer saline purgatives, diuretics and diaphoretics to promote absorption of fluid. When the œdema most affects the legs, as in the cardiac and hepatic forms, the foot of the bed should be raised. Bandaging the legs in such cases, if carefully done, makes the patient more comfortable, and may stay the progress of the swelling. In renal œdema a salt-free diet is occasionally successful in causing its absorption when other measures have failed. When œdema is considerable and persistent the subcutaneous tissues may be drained by inserting Southey's tubes, one or two, in each leg, or by making six or eight punctures in each with a tenotome. Since the skin is thin, shiny, and badly nourished, and also becomes sodden by the leaking fluid, the strictest aseptic precautions should be observed. When drainage is employed the head of the bed should be raised. Aspiration of a hydrothorax or an ascites may be necessary. In toxic œdema treatment should be initiated by a dose of castor oil and by irrigation of the bowel with a weak

alkaline or antiseptic solution. If the motions remain offensive, creosote ($\frac{1}{2}$ min.) may be added to a castor-oil mixture containing 10 min. of the oil and administered three times daily. When œdema occurs in wasting diseases it is an indication of the urgent need for stimulation and warmth. Alcohol is particularly called for. The primary disease requires, of course, its appropriate treatment. In the more chronic and less common form which occurs in carbohydrate dyspepsia in infants, albumen water only should be given for twenty-four hours and then replaced by a diet rich in protein but relatively poor in carbohydrate, such as protein-milk, or whole milk. For œdema and sclero-œdema neonatorum warmth is essential; the infant may be kept in an incubator or, better, between blankets in a cot warmed by hot-water bottles or an electric bulb. To promote expansion of the lungs, respiration may be stimulated by a mustard bath and by attempts to induce crying, such as flicking with a towel.

LOCAL CŒDEMA

Varieties. 1. **Inflammatory œdema** needs no special description. It is commonly seen in the neighbourhood of wounds and infected areas, and is accompanied by other evidences of inflammation. Very similar is the œdema accompanying gouty arthritis.

2. Among the causes which produce **obstruction to the flow of blood in peripheral veins** are thrombosis and varicosity of the veins and pressure upon them. The pressure may be due to fracture, dislocation, foreign bodies, callus formation, to badly applied bandages or splints, to glands, new growths, or aneurysm. Pregnancy and pelvic tumours must be remembered in connexion with œdema of the legs.

3. **Obstruction to lymphatics** causes a form of white, solid œdema which can only be pitted with considerable difficulty or not at all. It is met with in elephantiasis, and when the lymph-channels are subjected to pressure or involved in scar-formation. The white leg of the puerperium (*phlegmasia alba dolens*) is probably due partly to infection and partly to venous and lymphatic obstruction.

4. (Edema is included among the **trophic disturbances** induced by lesions of the lower motor neurone. It is often a prominent feature of brachial neuritis.

5. **Angio-neurotic œdema** is an unexplained form of circumscribed œdema which resembles urticaria in many respects. It may be induced in certain persons by particular forms of food.

In some it may be strawberries, oranges, or lemons, in others eggs, in yet others porridge. J. G. Freeman has designated this group toxic idiopathy. In some patients there is no apparent connexion with food. Temperamentally these subjects of angio-neurotic œdema are often highly strung and nervous. Sometimes it is hereditary, and many cases have been described in the same family. Occasionally what appears to be a typical œdema of this form is associated with febrile disturbances and suggests a toxic origin. By many it is regarded as an anaphylactic phenomenon. The œdema usually occurs suddenly; it is generally without associated symptoms, and often affects the same area again and again. Common sites are the face, especially about the eyes, and the tongue. The swollen patch pits on pressure. Sometimes it is purplish in colour, resembling purpura. It often lasts only a few hours, but may persist for a day or two, and is only serious if it affects the larynx.

The treatment consists in avoidance of any ascertained cause, such as particular articles of diet, and in the administration of alkalis. In some cases general tonic treatment by iron and arsenic proves successful. Calcium lactate may be tried, but is often disappointing. If the larynx is affected, tracheotomy or intubation may be necessary (see LARYNX, (ŒDEMA OF)).

6. **Trophœdema.**—This term has been applied to a disorder of unknown origin, the characteristics of which are a firm œdema of one or more extremities, insidious in onset, and unaccompanied by constitutional disturbance. As a congenital, hereditary, and familial affection it was described by Milroy and is known as *Milroy's disease*. In many other instances the œdema has not dated from birth but has appeared at various ages, though it is seldom deferred long after puberty. The legs are more frequently affected than the arms. In some cases both legs and both arms are equally œdematous; in others the distribution of the œdema is irregular, in yet others it is confined to one limb. The œdema is worse when the patient is erect and, when slight, may disappear during the night or when he is confined to bed, to reappear when the erect position has been assumed for a few hours. The only symptoms are a sense of weight and occasionally throbbing and heat in the limb, with perhaps aching and discomfort after much walking or standing. Sometimes the œdema slowly progresses until the limb becomes very large and unwieldy (pseudo-elephantiasis).

Treatment.—Rest in bed or skilled massage

reduces the œdema, but it invariably returns to its original proportions when the treatment is discontinued. When the lower extremities are affected, firm support by elastic stockings or bandages relieves the patient's discomfort. No drug has been found useful.

7. With **purpura** (q.v.), especially Henoch's purpura, patches of transient œdema are sometimes met with.

8. **Tetany** is often accompanied by œdema of the backs of the hands and feet, whence it may spread to the arms and legs. Less commonly the face also is affected. Perhaps analogous is the œdema which occurs in the same positions during the initial stages of **rheumatoid arthritis**. In that disease it may precede any sign of joint involvement for several weeks. Possibly both have a common toxic origin.

9. Exposure to **extremes of heat and cold** may cause œdema in unprotected parts. When cold is accompanied by wetting, œdema is especially prone to develop. It occurs, therefore, in the hands in such occupations as bottle-washing, and in the feet in trench warfare.

For œdema of the tongue, see TONGUE, ACUTE ŒDEMA OF. FREDERICK LANGMEAD.

ŒDEMA, HEREDITARY (see ŒDEMA).

ŒDEMA NEONATORUM (see ŒDEMA).

ŒDEMA OF GLOTTIS (see LARYNX, (ŒDEMA OF)).

ŒDEMA OF LUNG (see LUNG, (ŒDEMA OF)).

ŒSOPHAGOSCOPY AND GASTROSCOPY.—**Œsophagoscopy**, or the examination of the œsophagus by direct vision, has only taken its place as a routine method during the present century, as a result of the practice and teaching of Killian, von Eicken, and Brünings, who have established the importance of endoscopic methods in the diagnosis and treatment of disease in the upper air and food passages.

The apparatus used consists of a handle, at the upper end of which is an electric lamp, and lens. The light, impinging on a slotted or perforated mirror set at an angle of 45° to the beam, is reflected down tubes of varying length and size, the examiner viewing through a slot or perforation, and thus getting a direct view of growths, or foreign bodies, or the normal mucosa of the area under examination.

The patient may be examined in the supine position, flat on the table, with the head

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resting on a small pillow; this is the rule under a general anæsthetic. Under cocaine the patient can be examined sitting on a low stool, or preferably sitting astride a chair, with arms folded over the back of it. The body should be bent forward and the head slightly extended.

The following is the method of examination under local anæsthesia with the patient in the

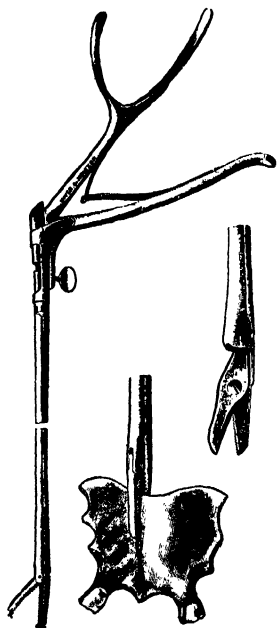


Fig. 66.—Irwin Moore's cutting forceps.

sitting position: A hypodermic injection of morphia hydrochloride $\frac{1}{4}$ gr. and atropine sulphate $\frac{1}{16}$ gr. having been given half an hour previously, to reduce the secretion of saliva and to render the patient more tolerant, the lips, gums, palate, fauces, and deep pharynx are painted with a 10-per-cent. solution of cocaine to which a little adrenalin hydrochloride (1 in 1,000) has been added. Special care must be taken to paint effectively the epiglottis and arytenoid cartilages. The efficiency of the anæsthesia should be tested by passing the index finger into the deep pharynx; if no coughing ensues the examination may be proceeded with.

The tube is first passed directly backwards towards the tip of the uvula; and then the point depressed slightly until it impinges on the base of the tongue. This passed over, the posterior pharyngeal wall is reached, when on further depressing the point and passing it deeper the epiglottis is brought into view. By passing the tube down behind it, at the same time hooking it forward, and then bringing the tube into a more vertical position, the arytenoids are seen.

Directly behind and between the arytenoids is a slit, situated between the two pyriform fossæ. The tube can be made to open up this slit or pass into either fossa, bringing into view the deep pharynx.

The displacement of the cricoid ring forward discloses the mouth of the œsophagus, which may be either a rosette-shaped dimple or a small hole; it may alternately open and shut with respiration. After the tube has been passed well into the cervical œsophagus, the lumen of the gullet appears open, expanding during inspiration and partially collapsing during expiration.

From the deep pharynx onwards the œsophagus inclines backwards and slightly to the left until it reaches the opening in the diaphragm (an oblique slit), to become continuous, $\frac{1}{2}$ –1 in. lower, with the stomach.

A great deal of the success of œsophagoscopy depends on never passing the tube (or any instrument introduced) onwards except under vision. Adhere strictly to the middle line, as otherwise a pyriform fossa may be entered and perhaps torn; or even perforation of the wall of the œsophagus may occur.

By means of œsophagoscopy foreign bodies, such as coins, pins, fish, rabbit or meat bones, or masses of meat, can be seen and removed with suitably shaped forceps. If the foreign body is too large to be removed up the tube, it is pulled up to the tip of it, and tube, forceps, and foreign body removed *en bloc*. This procedure has entirely superseded the old probang, which has to be passed blindly, and is most unscientific in action.

In the case of tooth-plates, or open safety-pins, it may be necessary to cut them into two or more pieces. This can be done with a pair of specially devised cutting forceps designed by Irwin Moore, each portion of the plate or pin being removed separately. (Fig. 66.)

With the aid of œsophagoscopy, too, papillomata can be snared; simple and malignant strictures examined and dilated by means of

ŒSOPHAGUS, AFFECTIONS OF

graduated bougies; portions of growth removed for microscopical examination; and intubation tubes passed for feeding purposes, thus obviating gastrostomy in many cases. Radium can be applied to cases definitely found to be malignant in character, often with most gratifying results.

The best intubation tube (Hill's pattern) consists of a small rubber tube 50-60 cm. long, mounted on a vulcanite point at the end of a thin piece of silver wire. The wire lies in the rubber tube, giving it stability, and prevents its being coughed out; and a small hole cut just above the vulcanite end permits food poured in at the top through a small funnel to emerge into the œsophagus below the stricture. The free upper end is strapped on to the cheek. This tube can be worn for weeks at a time.

Gastroscopy.—Ocular inspection of the gastric mucosa is seldom necessary, and, moreover, gastroscopy, except in the hands of an expert, is a procedure more dangerous than an exploratory laparotomy. The only method which conforms to sound endoscopic principles is that of combined direct and indirect œsophago-gastroscopy devised by Hill and Herschell in this country, and independently by Foramotti in Vienna. An extra-long straight œsophagoscopic tube is passed under direct vision into the stomach, as a guide for the insertion of a straight periscopic optical apparatus, with a lateral window on the principle of the cystoscope. There is also an arrangement for inflating the stomach to facilitate inspection. The technique closely resembles that of deep œsophagoscopy as regards anaesthesia and position. Ulcers, bleeding-points, and malignant growths may be made out by this method in a certain percentage of the cases examined.

CHARLES W. M. HOPE.

ŒSOPHAGUS, AFFECTIONS OF. —

Under this title are included:

1. ŒSOPHAGITIS—Ulceration.
2. ŒSOPHAGEAL OBSTRUCTION.
 - Scarring.
 - Carcinoma.
 - Diverticula.
 - Dilatation.
 - Spasm.
3. ŒSOPHAGEAL RUPTURE.

1. ŒSOPHAGITIS

Acute inflammation of the œsophagus may arise spontaneously, or as a symptom of general disease, or as the result of trauma.

(1) Spontaneous œsophagitis is uncommon.

Two forms are recognized—(a) an acute inflammation occurring in suckling infants without any discoverable cause, and (b) acute phlegmonous œsophagitis. This latter occurs in adults, and consists of a local or general swelling of the mucous membrane with purulent infiltration of the submucous tissue. The causal organism is probably a streptococcus. The symptoms are fever, and pain which becomes intense on swallowing; the condition is usually rapidly fatal. Owing to its rarity, the exact diagnosis is seldom made during life. Treatment is difficult; it is impossible to influence the progress of the disease by local measures, but cocaine lozenges may be necessary to give relief. Leeches to the neck are useful when the upper end of the œsophagus is involved.

(2) Symptomatic œsophagitis is not uncommon; it is seen occasionally in most of the acute febrile diseases, e.g. diphtheria, enteric, smallpox. In these the condition is often masked by the severity of the general symptoms.

Membranous œsophagitis is seen in several diseases. As a rare phenomenon, a true diphtheritic membrane occurs. More commonly the exudate is fibrinous; it may accompany pyæmic conditions and pneumonia.

Thrush may occur in the upper part of the tube, and must be remembered when a white membrane is found in this situation in wasted infants.

In hysteria a fibrinous cast of the whole or part of the œsophagus has been known to form.

The pain of symptomatic œsophagitis is slighter than that of the spontaneous phlegmonous variety.

Local inflammation may arise round an impacted foreign body, or by extension from an inflammatory focus in the vicinity, e.g. mediastinal abscess.

(3) Traumatism.—The swallowing of corrosives is followed by acute inflammatory reaction, which is most severe at the natural narrowings of the lumen of the tube, viz. at its commencement, at the termination, and at a point 4 in. along its length.

Ulceration is not uncommon. It is most often the result of injury from a foreign body, or the swallowing of corrosives. Naturally, ulceration is most likely to occur in those situations where the lumen is narrowest. The symptoms are pain on swallowing, or inability to swallow. Tuberculous ulceration is some-

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times seen as the result of swallowing tuberculous sputum. Syphilitic ulceration may be produced in the secondary or tertiary stages of the disease, but is rare. Ulceration from the pressure of an aortic aneurysm is occasionally seen and leads to slight repeated hæmorrhages, followed after a few days or weeks, when the aneurysm ruptures, by one which is profuse and fatal.

2. OBSTRUCTION OF THE ŒSOPHAGUS

Œsophageal obstruction may be divided for descriptive purposes into that due (a) to changes outside the œsophagus, (b) to changes in the œsophagus itself.

The **symptoms** of both groups are similar—a difficulty in swallowing, accompanied usually by the feeling that the food “sticks” somewhere in its œsophageal course, and regurgitation; pain is a variable symptom, but is rarely severe. If the obstruction be considerable and has existed for some time, the œsophagus may undergo dilatation above the level of the obstruction, and food may accumulate in the dilated portion. Regurgitation may thus be delayed for some hours and fermentative changes occur. In such circumstances there may be some difficulty in deciding whether the food has been returned from the stomach or from the œsophagus. Two features denote that it failed to reach the stomach—(a) its alkaline or neutral reaction, (b) the absence of peptone, showing that no gastric digestion has taken place.

The **diagnosis** of œsophageal obstruction may be made with certainty by (1) œsophagoscopy, (2) bismuth-feeding and X-rays, (3) passing a bougie, (4) auscultation.

(1) The *œsophagoscope* affords visual evidence of the obstruction and may disclose its nature, but the method is one which requires no small degree of practical skill and is not without danger (see (ŒSOPHAGOSCOPY AND GASTROSCOPY)).

(2) *Bismuth-feeding* is harmless and of great assistance. The patient, while in the upright position behind a fluorescent screen, is given a semi-solid mixture such as porridge or gruel which contains an ounce of bismuth oxychloride. A more satisfactory “meal” consists of barium sulphate (120 gr., 8 gm.) intimately mixed with a teaspoonful of bread and milk. Bismuth salts, with the exception of the oxychloride, are not devoid of risk, whilst barium sulphate is quite inert, as well as less expensive. If an obstruction be present the bismuth is

seen as an opaque mass “held up” at the stricture, or possibly passing on through it in a thin stream. (PLATE 45, Fig. 5, Vol. III, facing p. 556.) This method, besides confirming the presence of an obstruction, is helpful in estimating its degree and its length.

(3) The use of the *sound* remains the method of most general applicability, in spite of its danger, and of its small degree of usefulness in indicating the nature or extent of the obstruction. It is important to use a sound which is warm, rather flexible, and well lubricated, and to employ the minimum of force in passing it onward. It is well to try to pass a sound of medium size first, for this is less likely than a small one to set up spasm, or to cause perforation of the œsophagus. Before passing the sound, a careful examination should be made to exclude the presence of an aneurysm.

The most useful type of sound is that consisting of a thin flexible steel ribbon terminating in a screw to which olivary “heads” of various sizes can be attached. A head can usually be found which will pass through the stricture. The distance between the level of obstruction to ingress and egress indicates the length of the stricture. Before withdrawing the sound, mark upon it the point in contact with the incisor teeth; it is then possible to measure exactly the situation of the obstruction.

(4) *Auscultation*.—The entry of solid food into the stomach is audible if the stethoscope be applied to the epigastrium or 10th dorsal spine. The sound (the “swallowing murmur”) is heard normally six seconds after the bolus leaves the mouth, and any delay in transmission through the œsophagus may be disclosed by a delay in the production of this murmur. Though not conclusive, this test is of some value.

The **causes** of obstruction and the appropriate methods of **treatment** may be considered under the following headings:

(1) **Scarring** is most apt to produce obstruction at one of the three narrowest parts of the tube—at its commencement, at the fourth inch, or at its termination. The *diagnosis* is suggested by the history of the case. *Treatment* consists in gradual dilatation by the daily passage of bougies, or the frequent insertion of a Symonds tube for a period of a few days.

(2) **Carcinoma** has a tendency to occur at the narrow portions, but is most common in the lower third of the œsophagus. The condition is seen chiefly in men of 40–50 years of age, and its onset is insidious. Pain is a variable

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symptom. The *diagnosis* is made by the methods outlined above, by the extreme wasting, and occasionally by the presence of blood or of cancer-cells upon the bougie. Even in an early stage secondary involvement with enlargement of the left supraclavicular lymphatic glands may occur. The *dangers* of the condition are—progressive asthenia, the usual cause of death; perforation of the œsophagus into the pleura or mediastinum, causing empyema or abscess, and hæmorrhage from erosion of a vessel.

Treatment.—Local excision is very rarely possible. Gastrostomy is a valuable measure for preserving nutrition, allaying thirst, and relieving the pain when ulceration has occurred. When the diagnosis is certain, gastrostomy should be performed without delay.

(3) **Diverticula** are of two varieties—(a) congenital pouches, (b) traction diverticula due to pericœsophageal scarring as the result of cicatrization of a mass of bronchial glands.

Congenital pouches occur at the upper end of the tube, and are really pharyngeal in origin. Sometimes the œsophagus ends blindly at the level of the bronchus, with which the lower end communicates directly. More commonly, a local bulging only, at the upper end of the œsophagus, is present; it arises from the posterior wall, and may give rise to an intermittent swelling in the neck.

Traction diverticula are formed at about the level of the bronchial glands.

The *diagnosis* of diverticula rests upon intermittent difficulty in swallowing due to interference by the pouch with the lumen of the œsophagus, the presence of an intermittent swelling in the neck relieved by the regurgitation of undigested food, and the appearance under X-ray and œsophagoscopy examination. Successful surgical occlusion is occasionally possible.

(4) **Dilatation** as distinct from diverticula is occasionally seen. It may be uniform or local. The former variety occurs as a rare congenital abnormality, or as an acute condition analogous to acute gastric dilatation.

Local dilatations are liable to form above any œsophageal constriction and may be considerable in degree. Hypertrophy of the muscular coats of the tube is sometimes present. Symptoms of œsophageal obstruction accompanied by the regurgitation of a large amount of material from time to time establish the diagnosis.

Formerly included under this term were

the cases of nervous inco-ordination of the longitudinal and circular muscle-fibres of the œsophagus in which orderly opening of the cardia is prevented and obstruction occurs without hypertrophy of the circular muscle at the cardiac end of the œsophagus (achalasia of Hurst).

(5) **Spasm** of the œsophagus occurs in nervous children, neurotic adults, tabes dorsalis, and some forms of gastric disease.

In children of a nervous temperament, inability to swallow is a frequent complaint, and it may have reference to certain forms of food only, e.g. meat. The difficulty is psychical, but none the less real. It yields readily to "suggestion," and, if no notice be taken of it, will usually cease as suddenly as it commenced.

A somewhat similar difficulty is met with occasionally in adults, but is more resistant to treatment. Impressive treatments are advocated, and the best cure is by "suggestion." Starvation should not be resorted to, for the patients often take a delight in it, and after a few days lose all desire for food.

Œsophageal spasm in tabes dorsalis is uncommon, and fortunately the difficulty is intermittent. No form of treatment can be relied upon, though cocaine lozenges are sometimes useful.

"Cardiospasm" is a condition of spasm of the sphincter fibres of the lower end of the œsophagus, which are frequently greatly hypertrophied. The condition sometimes dates from early infancy, and may cause regurgitation of food from the beginning of life. Enormous dilatation of the œsophagus may ensue. A long history of regurgitation, tightness in the chest, and "asthma"-like attacks after meals, with perhaps faintness, is sometimes obtainable. The *diagnosis* is made by the method described above. *Treatment* consists in the free use of sedatives and alkalis. The insertion of a weighted tube (i.e. a blind rubber tube containing mercury) into the œsophagus daily is sometimes successful in otherwise obstinate cases. The spasm gradually relaxes and allows the tube to slip into the cardia.

3. RUPTURE OF THE ŒSOPHAGUS

Œsophageal rupture is extremely rare, except as the result of post-mortem decomposition. Rupture of the healthy tube has been known to occur from prolonged vomiting. Occasionally rupture has complicated diseased conditions of the œsophagus, such as neoplasms or ulceration.

C. E. SUNDALL.

OPERATIONS, AFTER-TREATMENT OF

ŒSOPHAGUS, FOREIGN BODIES IN
(see FOREIGN BODIES IN THE ALIMENTARY CANAL).

OLFACTORY SENSE, DISORDERS OF
(see SMELL, DISTURBANCES OF).

OLIGURIA (see URINE, VARIATIONS IN AMOUNT OF).

OMODYNIA (see MYALGIA).

OMPHALITIS (see UMBILICAL INFECTION IN THE NEW-BORN).

ONYCHIA (see NAILS, DISEASES OF).

OÖPHORITIS (see SALPINGO-OÖPHORITIS).

OPACITIES, DEGENERATIVE (see VITREOUS, AFFECTIONS OF).

OPERATIONS, AFTER-TREATMENT OF.—The after-treatment of a patient who has been operated upon depends on the nature of the operation and the patient's general condition.

In a healthy patient some operations cause so little inconvenience, beyond the necessity of having to stay in bed for a shorter or longer time, that it may be said that no after-treatment is necessary except keeping the bowels active and giving a light diet for a day or two. In severe or prolonged operations and in feeble patients many conditions require attention.

1. **Pain.**—This varies very considerably; it may be in or near the wound, or in a region quite apart from the site of the operation. Pain in the lumbar region is a common cause of discomfort. It is best prevented by placing a firm pillow in that area during the operation if it is likely to be a prolonged one, or by seeing that the patient's back is flat on the table. The pillow must not be so large as to increase the convexity forwards of the lumbar spine, which should lie on the pillow comfortably and lazily. When the patient is put back to bed a firm, flat mattress and a small pillow under the lumbar spine give most relief. An india-rubber hot-water bottle well wrapped in flannel and placed under the back, and a change in position, are also often useful. Now that patients are placed in a more or less upright position, with the knees flexed over a bolster, after the immediate effects of the anæsthetic have passed, this pain does not seem to be so pronounced a symptom. Pain in the wound may be slight or severe, inter-

mittent or constant, and is increased by such actions as movement, coughing, or vomiting. It is in part due to the stitches, especially if these are tied too tightly and are of unyielding material, such as silkworm-gut. It seems to be an earlier and more pronounced symptom when the patient has been anæsthetized with gas and oxygen, the anæsthesia passing off rapidly and the patient in consequence becoming quickly conscious of the pain. It is well, therefore, to infiltrate the operation area, especially if the abdomen is the site of operation, with a 1- or 2-per-cent. sterilized novocain solution, before making the incision. The novocain should be injected into the different layers; it certainly diminishes the after-pain.

Aspirin (15 gr.) and potassium bromide (20 gr.), given per rectum in saline solution, are frequently quite sufficient to relieve pain. The aspirin should be broken into a fine powder, and the combination of drugs repeated if necessary. Heroin ($\frac{1}{2}$ or $\frac{1}{4}$ gr.) given hypodermically is also useful. The routine use of morphia is to be deprecated: when it is required, $\frac{1}{4}$ or $\frac{1}{2}$ gr. is often sufficient. It is better to give smaller doses and repeat them if necessary than to give a larger one, especially in abdominal operations, when flatulence is so liable to cause severe pain for a day or two after the operation. The pain of flatulence is best treated by a turpentine enema (1 oz. to a pint of soap and water) or a purgative.

2. **Postanæsthetic vomiting**, when severe, may be treated by gastric lavage, using a pint of hot water containing a teaspoonful of bicarbonate of soda. Alternatively, if this solution be drunk quickly, it will often stop the vomiting. The dose may be repeated from time to time. Chlorotone in 5-gr. cachets is sometimes useful.

3. **Taste and smell of the anæsthetic.**—The patient may complain of the persisting taste of the anæsthetic. Slices of lemon to suck, or mouth-washes of glycothymoline or listerine, are valuable means of getting rid of the taste. Glycothymoline and listerine are most efficacious if applied all over the mouth by means of small swabs held in forceps. Occasionally the taste of the anæsthetic remains until the bowels are opened by a purgative.

4. **Hygiene of the mouth.**—Particular attention should be paid to the condition of the mouth, especially after abdominal operations and those involving the buccal cavity. It is not uncommon for the tongue to be slightly coated for a day or two after any operation,

OPERATIONS, AFTER-TREATMENT OF

but this quickly disappears when the bowels are opened. A clean, moist tongue is a good sign. When it remains furred and the patient is otherwise doing well, means must be taken to clean it. In the first place, no food must be allowed to remain in the mouth; after each meal the mouth should be cleansed by means of small dossils of wool or gauze soaked in some application and held in forceps. The teeth, tongue and gums are then successively gently rubbed over. Useful applications for this purpose are sodium bicarbonate solution (10 gr. to 1 oz.), glycerin and lemon, listerine, and carbolic-acid lotion (1 in 80 or 1 in 100). It may even be necessary to scrape the tongue with the handle of a spoon. In less severe cases the use of a tooth-brush and a mouth-wash twice a day are sufficient.

5. Position of the patient.—When the effects of the anæsthetic and the immediate shock of the operation have passed off, the position of the patient in bed has to be decided upon. In abdominal operations, in mouth operations, and in those on the neck the upright position should be adopted; the patient very quickly becomes accustomed to it, and is able to sleep quite comfortably. The mattress must be firm and not allowed to sag; fracture boards should be used, when necessary, to prevent sagging. An air ring under the buttocks is a source of much comfort. A firm pillow or bolster should be placed under the thighs so as to flex the knees, the ends of the pillow or bolster case being tied to the sides of the bedstead, and the lower end of the bed raised on 6-in. or 8-in. blocks to prevent the patient from slipping down in bed. Several pillows supported on a bed-rest or a special frame should be used to prop the back, shoulders and head; there should be no space or "hollow" between the patient's spine and the pillows. In such an operation as amputation of the breast, the arm on the affected side should be placed and fixed at a right angle to the trunk, the forearm being left free and lying on pillows. After an amputation the bedclothes should be so arranged over a cradle that the end of the stump is visible, and a tourniquet should be kept near at hand, in view of possible hæmorrhage. When the stump is exposed the bleeding at once becomes evident.

6. Shock and collapse.—In these conditions the most important factor is the lowered blood-pressure, and therefore, whatever the cause or combination of causes leading to them,

treatment is directed to raising and maintaining the blood-pressure. The lower end of the bed should be raised to a height of 12-18 in. and warmth applied to the extremities and trunk by means of hot bottles; the head may be wrapped in a warm flannel and hot bottles placed near. A simple method of maintaining warmth, and one that can often be used in private houses, is to raise the bedclothes by a cradle or old box, or any other convenient apparatus, and hang an electric-light bulb inside the space. With a little care, danger of burning the patient or the bedclothes can be avoided. The heat of the bulb soon raises the temperature under the clothes and maintains a constant supply of warmth for as long as necessary. The administration of saline solution into the axilla, or per rectum, or intravenously may be necessary. Which of the three methods is used depends to some extent on the degree of the shock and collapse. The introduction of the saline into the *axilla* is the simplest, and the fluid is quickly absorbed. A needle with a bore of 2 mm. is a useful size; it should be inserted just below the anterior axillary fold and directed upwards and inwards. In an adult a pint to a pint and a half can easily be run in without danger of causing sloughing of the skin and fat; in children a few ounces (4-8) may be introduced. The tenseness of the swelling is the best indication when to stop the flow. The patient may complain of a certain amount of pain afterwards. The same axilla may be used again.

The *rectal method* is convenient for continuous infusion. The flow should be so regulated that about a pint per hour is injected, the liquid in the reservoir being kept at a temperature of 105°-110° F., so that it will enter the rectum at about 100° F. After three or four pints have been administered it is generally advisable to stop the flow for two or three hours. An ounce of glucose may be added to each pint of the saline solution.

If the *intravenous method* is used, it is essential not to let the fluid run too quickly; a needle with a bore of 1 mm. is a convenient size. The rate should be about one pint in fifteen minutes, and not more than three or four pints should be injected at one time. It is better to repeat the infusion than to give massive doses which might produce œdema of the lungs. Recent experiences show that better results may be obtained by using a solution of gum arabic, 5 or 6 per cent., in isotonic saline solution, and the possibility of

OPERATIONS, PREPARATION FOR

using blood from another individual, especially when the shock and collapse is due to hæmorrhage, should be borne in mind (see TRANSFUSION). Pituitary extract ($\frac{1}{2}$ -1 c.c.) may be given hypodermically, and, when the patient is able to swallow, plenty of liquids, such as tea and hot water. (See also SHOCK AND COLLAPSE.)

7. General treatment.—Patients often complain of abdominal distension and discomfort from “wind,” even when, clinically, there is no evidence of distension of the abdomen. A turpentine enema (1 oz. to the pint), given by a rectal tube inserted as high as possible, is the best means of relieving this symptom. A soft rectal tube left *in situ* for some time often enables the flatus to pass. Pituitary extract ($\frac{1}{2}$ -1 c.c.) or eserine salicylate ($\frac{1}{10}$ - $\frac{1}{20}$ gr.), every four hours till four to six doses have been given, is sometimes beneficial. The bowels should be moved by a purgative—an ounce of castor oil is the most satisfactory one—usually on the morning of the third day. Subsequently, infusion of senna pods is one of the best means of keeping the bowels open.

Thirst is relieved by giving liquids and saline infusions. The juice of a lemon or orange, or acid drops, are grateful to the patient and are efficacious.

Retention of urine, particularly in women, after an abdominal operation is a common experience. If the patient is constantly passing small quantities of urine, the bladder should be carefully examined for distension, and a catheter passed. The retention may continue for many days.

8. Rarer complications, such as parotitis, acute dilatation of the stomach, delayed anæsthetic poisoning, massive collapse of the lungs, etc., will all require special treatment, as described in other articles. T. P. LEGG.

OPERATIONS, PREPARATION FOR.

—In a short article it is impossible to discuss in detail the question of preparation for operation, and each surgeon has his own particular method. Moreover the preparation, to some extent, varies with the precise nature of the operation, and modifications of it may therefore be necessary.

General preparation.—In hospitals and in modern nursing homes operating theatres are provided; it is only necessary, therefore, to consider the preparation of a room in a private *garage*. The room selected should be a well-lighted one, preferably with a northerly aspect,

and with a window near to which the operating table can conveniently be placed. The room should be capable of being warmed to a temperature of 70°-75° F. All unnecessary furniture should be removed, and, if time permits, the carpet should be taken up and the room swept and dusted. If, however, the operation has to be done at short notice, it is better not to stir up the dust, for it is that which gets into the wound that matters, and not that which remains outside. Most practitioners who operate have a portable operating table; if not, one can be hired from an instrument-maker. The operator should interview the nurse before she proceeds to the patient's house, and give his instructions as to dressings, preparation of the patient, provision of bowls, water, saline solution, and the lotions he will require. In most cases it is desirable for the patient to be in the nursing home, or the nurse to be in the house of the patient, for at least twenty-four hours before the operation is done, thus enabling the patient to become accustomed to the new surroundings and to become acquainted with those who will be in charge after the operation; it gives time, too, for the patient to be properly prepared. For some operations, such as gastro-enterostomy, and for old people, a longer time is desirable.

Except in grave emergencies the urine should be tested carefully, and, in the case of old people, the amount passed per diem, its specific gravity, and the amount of urea secreted should be determined, particularly if the operation is to be a severe one or on any part of the urinary tract. An estimation of the efficiency of the renal function should be made. In renal operations it is always desirable to know that the functions of each kidney are efficient, especially when nephrectomy is contemplated. By collecting the urines separately through ureteral catheters the functional efficiency of each kidney can be estimated. In operations on the prostate and bladder the prognosis is very considerably influenced by the state of the renal function. While the chemical tests of renal efficiency are valuable, they must always be considered in relation to the clinical signs: severe thirst, a dry tongue, a rapid pulse, and persistent vomiting suggest impaired renal function. The chemical tests may suggest an efficient renal function, but the clinical signs may not be in accord with such findings. There are many different methods of estimating renal efficiency (see p. 339). In the present state of our knowledge the clinical

OPERATIONS, PREPARATION FOR

evidences of the functional efficiency of the kidneys are of as much importance as those obtained by chemical tests; the latter have especial value when the urine of each kidney is separately tested. The presence of sugar is of great importance; the choice of the anæsthetic, the necessity of dieting before and after the operation, and of administering sodium bicarbonate by the mouth or intravenously, have to be determined. When the patient is feeble, or shock is present or likely to arise during or after the operation, means must be taken to combat it, such as rectal, subcutaneous or intravenous infusions. Recently, transfusion of blood has been employed; whether the method will become widely available in ordinary practice remains to be seen, but the introduction of the citration method increases its possibilities. It is the general custom nowadays to inject morphia (or one of its modifications) and atropine from three-quarters to an hour before the administration of the anæsthetic is begun. After the hypodermic has been given the patient should be kept absolutely quiet in bed, and at the proper time lifted gently on to the table: he should not be allowed to walk to the table.

In all operations on or about the mouth and on the gastro-intestinal tract, special care should be taken to render the buccal cavity as free from germs as possible. Stumps and decayed teeth should be removed; tartar should be scraped away, and the teeth and gums carefully cleansed by means of swabs held in forceps. Bicarbonate-of-soda lotion (10 gr. to 1 oz.), followed by carbolio (1 in 80), is useful and efficient. Mouth-washes may also be employed, and the tongue should be cleansed, the handle of a spoon being used to remove the fur and coating.

For purgation before the operation, castor oil (1 oz.) is the best drug. It should be given on the afternoon or evening before the operation. Few patients refuse to take castor oil or profess to be unable to swallow it. Given in brandy or with lemon it is almost tasteless, and the last portion remaining in the mouth can be removed by eating a biscuit, brushing the teeth, or sucking a slice of lemon. After it has been administered, light, easily digested food only is permissible; the kind of food depends upon the nature of the operation, for a patient who is to have an operation on the stomach requires food different from one who is to have an operation on the kidney. As regards nourishment, it is now quite usual to

allow the patient to have tea or beef tea, about 4-6 oz., as late as a couple of hours before the operation is performed. A soap-and-water enema should always be given on the day of the operation, and, when the rectum is the site to be operated upon, more than one is usually required to complete the evacuation of that part of the bowel. In operations on the stomach it may be necessary to wash out the viscus completely; in that case nutrient enemata and salines per rectum are given.

Different methods of local preparation are in vogue. Iodine and picric acid are most frequently used, but I do not think that they are superior to the older antiseptic method. They have, perhaps, one advantage in being "dry" methods. Whatever method is used, the part should be well washed with soap and water and all extraneous dirt removed. Shaving the hair, as much as may be required, should never be omitted. Before the iodine (2 per cent in rectified spirit) is applied, the water and soap should be removed by washing the part with methylated spirit. If picric acid is used the operation area should be cleansed with soap and water, and a compress of a 1-per-cent. solution applied some hours before the operation, the parts then being treated with a 3-per-cent. solution of the acid in methylated spirit. The advantages of picric acid are that it penetrates more deeply into the tissues and does not cause desquamation of the skin. Some patients are very susceptible to iodine, and suffer severe pain and discomfort from the local effects of an acute dermatitis. Double cyanide gauze should never be employed after the operation as a dressing, if iodine has been used for the skin; it is not unlikely to produce an intense dermatitis which may be mistaken for erysipelas.

In operations on joints, especially the knee, where the skin is thick and rough, every care should, of course, be taken to cleanse the skin from dirt before the operation. This means that, except in acute suppuration, the preliminary treatment should extend over two or three days if the skin is dirty. Frequent washing and the use of turpentine or ether to remove the dirt should be employed before the antiseptic preoperative dressing is applied. After the dirt has been removed, the skin all round, as well as above and below the joint for some distance, should be washed with 1-in-20 carbolic, and then 1-in-2,000 perchloride of mercury should be applied for some hours. If iodine is employed, the same careful cleansing

of the skin by soap and water and turpentine or ether should be carried out, and the iodine allowed to dry before being covered with sterilized gauze. At the operation a fresh coat of iodine is applied all round the joint. The same precautions must be taken if picric acid is used. The "carbolic method" is, in my opinion, the best, and I can only condemn the perfunctory application of iodine, which is not infrequently employed only just before the joint is opened.

T. P. LEGG.

OPHTHALMIA NEONATORUM (*see* CONJUNCTIVITIS).

OPHTHALMIA, SYMPATHETIC (*see* UVEAL TRACT, AFFECTIONS OF).

OPHTHALMOPLÉGIA.—Paralysis of the ocular muscles may be due to lesions of the motor nerves that supply them—that is, the third, fourth, and sixth cranial nerves—or of the nuclei from which these nerves spring. Any one of the muscles may be paralysed alone, or two or more may suffer at the same time; this is likely to be the case when the third nerve, which innervates several of the external ocular muscles, is affected, or when more than one nerve is involved by disease.

The **symptoms of ocular palsy** are *limitation of the movement of the eye in the direction in which it is normally rotated by the paretic muscle, and consequent squint or strabismus*, but this is visible only when the patient looks in a direction that would require the action of the affected muscle. As a result of the squint or non-parallelism of the visual axes, any object that forms an image at the macula of the normal eye also throws an image on some other non-corresponding part of the retina of the other eye, and the patient consequently projects two images of that object externally, and therefore sees double or has *diplopia*. Further, on attempting to employ the weak muscle he misjudges the position in space of the object seen, or has *erroneous projection*; if then he tries to touch the object his finger goes in a wrong direction, the error being always in the direction of action of the affected muscle. Diplopia and erroneous projection are always the most troublesome symptoms of an ocular palsy.

It is by a study of the diplopia that we can determine which muscle or muscles are affected if the palsy is not obvious on movement. The rules that guide us are these: When an abductor muscle is weak, and there is consequently

a convergent squint, the diplopia is homonymous—that is, the image seen by the right eye is to the right of that seen by the left; while in a divergent squint due to weakness of an adductor the diplopia is crossed. Similarly, if there is paresis of an elevator muscle the image seen by the affected eye is higher than that of the normal, and when a depressor is feeble the image is projected too far downwards. In the second place, the images separate farther on looking in that direction in which the weak muscle comes into action; and finally, when the images are side by side and the diplopia is homonymous, the false image belongs to the eye of that side towards which movement increases the distance between the images, or, if the diplopia is crossed, to the other eye.

Paralysis of the third nerve may result from lesions affecting any part of the nerve or its nucleus. The most common cause of a nerve lesion is basal meningitis, especially syphilitic meningitis, as the interpeduncular space through which the nerve passes is one of the commonest sites of this disease. Injury to the skull, particularly if the base is fractured, often produces either a partial or a total palsy which is frequently transient; it may be due either to direct injury of the nerve or to hæmorrhage into its sheath. The nerve may also be damaged by thrombosis of the cavernous sinus, compressed by aneurysm or dilatations of the carotid artery, involved in an inflammatory process or growth in the orbit, or in periostitis of the sphenoidal fissure. A nuclear palsy may result from hæmorrhage or tumour in, or compression of, the upper part of the midbrain, or from inflammatory lesions here. The most common of the latter are polioencephalitis superior, which occurs in chronic alcoholism, and lethargic encephalitis, which has a predilection for the grey matter of the midbrain. There is also a form of progressive nuclear ophthalmoplegia which involves the third as well as the other ocular nuclei; its pathology is a chronic degeneration of the cells of the nuclei. A form of recurrent ocular palsy which involves most commonly the third nerve is not uncommon; as it is generally associated with unilateral headache it has been termed *ophthalmoplegic migraine*. Recovery from the first few attacks is usually complete, but after repeated attacks some of the muscles are likely to become permanently paralysed. Finally, third-nerve palsy is common in certain nervous diseases, particularly in tabes and disseminated sclerosis. In tabes some or all of the muscles supplied by

OPHTHALMOPLÉGIA

the nerve may be weak, or the palsy may be total and complete; it may be due to an associated chronic or gummatous meningitis that involves the trunk, or to a nuclear degeneration. The former is probably the cause of the temporary palsies that occur in this disease. In disseminated sclerosis the paralysis is generally partial and slight, and often transient.

The *symptoms* depend on whether the palsy is partial or complete. When it is complete, all the external ocular muscles are paralysed, except the external rectus and the superior oblique, and the only movements that remain possible are outward deviation by the former, and downward and outward rotation by the latter. As the levator palpebrarum is also powerless, there is ptosis and the lid cannot be raised. The internal ocular muscles, too, are paralysed; the pupil is dilated and fails to contract when light is thrown into the eye or on attempting to converge, and the power of accommodation is abolished. In partial palsies certain only of the external or intrinsic muscles may be affected, or some or all of them may be merely weak. Occasionally only the external muscles are involved, or the intrinsic alone may suffer; this local internal or external ophthalmoplegia is more common when the disease is limited to the nucleus.

Iridoplegia, or palsy of the iris, becomes obvious in disturbances of its reflex action. The light reflex is obtained by throwing light into, or suddenly uncovering one eye in a bright room. The pupil of this eye contracts at once, and normally remains contracted—the direct reflex; and if the nervous connexions are normal the pupil of the other eye also becomes small—the consensual reflex. The path of this reflex is through the optic nerve, both optic tracts, probably the anterior quadrigeminal body, and the anterior part of the third nucleus, and a lesion in any of these parts may abolish it. It is absent in blindness due to retinal or optic-nerve disease, and when the third nerve is paralysed. But it occurs apart from blindness and ocular palsies, and is then probably due to an affection of the fibres that connect the anterior quadrigeminal body with the third nucleus. Then the pupil still contracts on accommodation. This isolated reflex inactivity to light is known as the *Argyll-Robertson sign*. It is very common in tabes and general paralysis, but occurs also in cerebro-spinal syphilis and, more rarely, in chronic alcoholism. Lesions of the anterior part of the midbrain also produce it.

Accommodation iridoplegia.—The pupil normally becomes smaller on accommodation, but this reflex or associated contraction is lost when the third nerve is paralysed or its nucleus damaged. Accommodation may be impossible too, but this is not always so; in tabes the pupil occasionally fails to contract, though the power of accommodation is preserved.

Cycloplegia, or loss of accommodation, also results from lesions of the third nerve or its nucleus, or of the ciliary ganglion or short ciliary nerves. It is most commonly seen as part of a third-nerve palsy, but it also occurs as an isolated phenomenon in diphtheritic palsy.

Trochlear palsy.—An isolated palsy of the trochlear or superior oblique muscle is rare. The fourth nerve, which innervates it, or its nucleus may be involved by tumours, hæmorrhages, or softenings in the region of the posterior quadrigeminal body, or the nerve by meningitis, trauma, and other lesions in its intracranial course, or by a neuritis secondary to a cellulitis or periostitis of the orbit.

The downward and outward movement of the eye is affected, and homonymous diplopia occurs on turning the eye in this direction, the false image being lower than, and its upper end tilted towards, the true. To avoid the inconvenience that the diplopia produces, the head is generally tilted forwards and towards the sound side.

External rectus palsy.—The sixth or abducens nerve is probably the most commonly affected, as its long intracranial course, from the lower margin of the pons to the sphenoidal fissure, exposes it to many pathological conditions. The nerve may be involved in pontine hæmorrhages, softenings, and tumours, and its extramedullary portion often suffers in meningitis, meningeal hæmorrhages, and basal fractures; it is frequently paralysed, too, when intracranial pressure is raised by cerebral tumours or other causes, as it is then compressed between brain and skull, or stretched owing to the backward displacement of the brain-stem. The symptoms are a convergent squint with defective outward movement of the affected eye and homonymous diplopia.

Combined ocular palsies.—More than one of the ocular nerves may be paralysed at the same time; this is particularly likely to happen when they are involved by disease at the base of the brain, or in the neighbourhood of the sphenoidal fissure, where they run close together. A common cause of this condition is basal meningitis, especially the *syphilitic*

OPHTHALMOPLÉGIA

variety. It also occurs as a result of nuclear disease, both in inflammatory affections as polioencephalitis and lethargic encephalitis, and in that primary degeneration of the nuclear cells which constitutes progressive nuclear ophthalmoplegia. It may be also due to orbital cellulitis and periostitis.

Conjugate ocular palsies.—Instead of paralysis of the individual muscles we may find the movements of both eyes limited or lost in one or more directions, but as the defect of movement is equal in the two eyes the optic axes remain parallel and diplopia does not occur.

The most common conjugate palsy is that of lateral movement. In the neighbourhood of each sixth nucleus, near the floor of the fourth ventricle, there is a centre that receives the voluntary impressions which excite lateral deviation of both eyes to the same side; from this supranuclear centre one system of fibres passes to the sixth nucleus on the same side, and another, probably through the dorsal longitudinal bundle, to that part of the third nucleus from which the fibres that supply the opposite internal rectus arise. A lesion of this supranuclear centre makes it impossible for the patient to turn his eyes towards the affected side, but the muscles themselves are not paralysed, as the internal rectus still contracts on accommodation.

Conjugate palsies of the upward and downward movements of the eyes are a frequent result of midbrain lesions, but the actual positions of their supranuclear centres have not been yet determined. The reflex contraction of the pupil to light is generally lost with conjugate paralysis of upward movement, and the power of accommodation with weakness of the conjugate downward movements. An associated loss of convergence and accommodation is sometimes due to more posterior lesions in the midbrain.

Treatment.—The treatment of ocular palsies is that of the conditions to which they are due. Syphilis, as the most common cause, should be always considered. Diplopia, which is the symptom that gives the patient most discomfort, can be controlled by wearing a shade over the affected eye. GORDON HOLMES.

OPHTHALMOSCOPY (*see* EYE, EXAMINATION OF).

OPIUM POISONING (*see* POISONS AND POISONING).

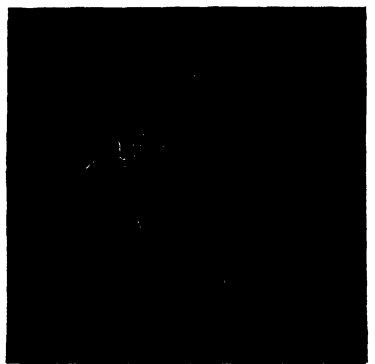
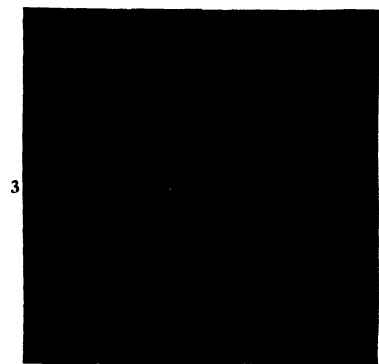
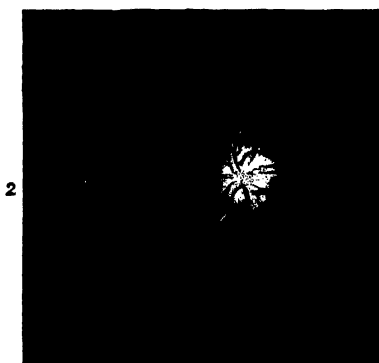
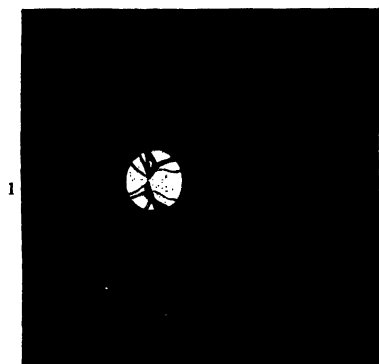
OPTIC ATROPHY

OPSONIC INDEX (*see* SEROLOGICAL DIAGNOSIS).

OPTIC ATROPHY.—The essential feature of optic atrophy is degeneration of the nerve-fibres in the optic nerve, as shown by increasing pallor of the disc and diminution of the visual functions. In optic neuritis and papilloedema the increased redness of the optic disc is due entirely to hyperæmia; the veins and capillaries are dilated and new capillaries form on the disc. But in optic atrophy the whitening of the nerve-head is due only in part to diminution in the size of the vessels, and in some cases is due mainly to replacement of normal nerve tissue by neuroglial tissue; there may be marked pallor of the disc before there is any sensible diminution in the blood supply.

Optic atrophies are commonly subdivided into two main classes—(1) primary, (2) secondary or consecutive (PLATE 23, Figs. 1, 2). A *primary* atrophy is defined as one in which the atrophic changes appear in the optic disc without any pre-existing inflammation or œdema of the disc. A *secondary* atrophy is one which follows on, or is consecutive to, a preceding papillitis or papilloedema. The subdivision is a misleading one, and should be abandoned, if for no other reason than that in many cases of atrophy following papilloedema with swelling and hæmorrhages the disc may later show no signs of the pre-existing condition. The edges of the disc may be clear-cut without disturbance of the retinal pigment, the lamina cribrosa may show up clearly at the bottom of the white disc and the vessels present no change beyond a simple diminution of calibre. Moreover, a papilloedema may exist and may leave traces after its subsidence, and yet the atrophy may not be the result of the papilloedema but a coincident effect of pressure exerted on the intracranial portion of the nerve, as by a pituitary tumour. Indeed, it is not unusual to see in such cases a papilloedema developing in an eye in which the optic atrophy is already present.

Logically the term primary atrophy ought to mean the condition resulting from the direct action of some destructive process (pressure, trauma, or toxin) on the nerve-fibres themselves, and secondary atrophy should denote that the degeneration of the nerve-fibres is the consequence of some previous process, such as inflammation or degeneration, taking place in some structure or structures on which the nerve-fibres are dependent. On



1, Primary optic atrophy. 2, Papilloedema subsiding into secondary optic atrophy.
3, Papilloedema with swelling of the disc, hæmorrhages and exudates. 4, Albuminuric
neuro-retinitis. 5, Diabetic retinitis. 6, Retinal arterio-sclerosis.

this basis the true primary atrophies would be those occurring in glaucoma, high myopia, tabes, and lead or arsenical poisoning, or when there is direct pressure on the nerve in any part of its course. The secondary atrophies would include the post-neuritic and post-papilloedematous forms, those following on retinal and choroidal inflammations and degenerations, and atrophies from vascular causes and retrobulbar inflammations. It would, however, be much better, as I have said, to abandon the use of the terms primary and secondary altogether, and to classify the optic atrophies on the basis of the place of action of the primary cause. On this principle we get four main classes :

1. Atrophy due to causes acting at the optic disc.
2. Atrophy of intra-ocular origin (ascending atrophy).
3. Atrophy of retro-ocular origin (descending atrophy).
4. Atrophy due to causes acting directly on the nerve-fibres.

Where a condition such as optic atrophy may result from many different causes, any attempt at a simplified classification is open to criticism. The one I have suggested does, however, enable us to group together those cases which are allied to one another in their causation.

1. This class, in which the atrophy is due to **causes acting at the disc itself**, includes cases consecutive to optic neuritis and papilloedema, cases of glaucoma, and a small number of cases occurring in high myopia. In optic neuritis and papilloedema a swelling and degeneration of nerve-fibres takes place, the neuroglial elements proliferate, and the newly formed connective tissue that results contracts down and constricts the remaining nerve and vascular elements in the disc. In this form of atrophy the disc has an opaque solid look and the vessels are diminished in calibre. The swelling usually invades the retinal tissues for some little distance round the disc, and the after-effect of this disturbance is to produce an eroded appearance of the retinal pigment at the edges. In glaucoma the increased intra-ocular pressure acts on the nerve-fibres, the lamina cribrosa, and the blood-vessels, and the yielding of the lamina cribrosa results in further disturbance of the nerve-fibres by stretching them over the more unyielding edge of the sclera. The result is a pure atrophy, with recession of the disc, and with the lamina cribrosa showing up

clearly at the bottom of the cup. An almost indistinguishable appearance is rarely seen in cases of high myopia.

2. **Atrophy of intra-ocular origin (ascending atrophy).**—The great majority of the nerve-fibres of the optic nerve arise from the ganglion cells of the retina. Retinal degenerations will consequently give rise to atrophy of these fibres. In its most typical manifestation the atrophy resulting from retinal degeneration produces a curious wavy appearance in the disc. These retinal degenerations may be primary, as in retinitis pigmentosa and amaurotic family idiocy; or secondary to retinal or choroidal inflammations, interference with the retinal blood supply as in embolism of the central artery, arterio-sclerosis, or spasm of retinal arteries as occurs in cases of quinine, aspidium-flix-mas and lead poisoning.

3. **Atrophy of retro-ocular origin (descending atrophy).**—The optic nerve is liable to direct injury in any part of its course, but the commonest form of trauma is a basal fracture of the skull tearing the nerve in the foramen. The rupture may be complete or partial. The evidences of rupture of the optic nerve are loss of vision with complete loss of reaction of the pupil to direct light but good consensual reaction. The obvious signs of atrophy of the disc do not show for several weeks. The disc becomes white all over, with the lamina cribrosa clear and no immediate diminution in the calibre of the vessels. Hæmorrhage into the nerve sheath without actual rupture may cause atrophy.

Pressure on the nerve will cause atrophy and may arise (a) from orbital tumours, either intra- or extraneural; (b) in the foramen, from bony thickening or from arterio-sclerosis of the ophthalmic artery; (c) the most important, from intracranial tumours pressing directly on the nerve. These may be either frontal, temporo-sphenoidal, pituitary, or third-ventricle tumours. But intracranial tumours may also indirectly cause pressure on the nerves or chiasma by leading to distension of the floor of the third ventricle. Aneurysm or sclerosis of the internal carotid, again, may be a cause of atrophy.

Inflammation.—Atrophy of the disc results from inflammation affecting the nerve in any part of its course. If the inflammation attacks the nerve immediately behind the bulb, i.e. after the central retinal vessels have entered the nerve, there is nearly always papillitis, and this may modify the subsequent appear-

OPTIC ATROPHY, FAMILIAL

ances of the atrophy. The neuritis may be (a) acute, axial or peripheral, and due to causes acting in the orbit, foramen, or intracranially; or (b) chronic, as the toxic amblyopias, due to alcohol, tobacco, etc. (There is considerable doubt whether tobacco amblyopia should not be included among the atrophies of intra-ocular origin, as there is some evidence that the primary changes produced by tobacco poisoning are retinal.) Disseminated sclerosis is one of the commonest causes of retrobulbar neuritis and the consequent atrophy, and probably Leber's familial atrophy should be included in this group, though Fisher has produced some evidence to show that the latter belongs to the group of pressure atrophies and may be caused by pituitary swelling.

4. Atrophy due to causes acting on the nerve-fibres primarily and directly.—In this class is included the very large number of cases of tabetic atrophy. The toxin of tabes seems to act directly on the nerve-fibres, killing them. In these cases the disc may become very pale and the visual loss be very pronounced without any interference with the retinal circulation. Later, when the retinal degeneration has advanced, there naturally follows a shrinking of the blood supply. The disc has a bluish tinge in its pallor and may show a slight cupping, with the pitting of the lamina cribrosa visible in its whole extent. Certain poisons, such as the arylarsenates, soamin, atoxyl, and other arsenical preparations and lead, also act directly on the nerve-fibres.

I have not attempted, in the space at my disposal, to do more than sketch the more important causes of optic atrophy, and there remains a considerable residuum of cases in which the etiological factor is indefinite or unknown.

The **diagnosis** of any case must depend not only on ophthalmoscopic examination and a full investigation of the visual functions, including the pupil reactions, the fields for white and for colours, and the light sense, but also on evidence from other disturbances, especially of the central nervous system; only then can any prognosis be given or treatment decided upon. The latter will be directed to the underlying causes.

LESLIE PATON.

OPTIC ATROPHY, FAMILIAL (*syn. Leber's Disease*).—This rare disease is characterized by a familial tendency, and the rapid onset of amblyopia due to deterioration of central vision in both eyes. It is almost in-

variably transmitted through the female, and affects predominantly males. The symptoms may appear at any time from childhood to the sixth or seventh decades, but they develop most commonly about the age of twenty; many cases, however, commence at the age of puberty, and in women at the menopause.

Symptoms.—The first indication of the disease is usually a mistiness of central vision in one eye, which increases rapidly until within a few weeks sight is lost or greatly reduced. The second eye is occasionally affected simultaneously, but more commonly a few days or weeks later. In many cases the loss of central vision is complete, but peripheral vision usually remains intact, though there is occasionally a concentric contraction of the fields too. It leads, very rarely, to total blindness of one eye. If the fundi are examined at the onset the discs may appear hyperæmic and swollen; later they become pale, especially on the temporal sides.

The condition usually becomes stationary and permanent within a few weeks, but a certain number of cases regain a fair amount of central vision.

Leber recognized that this form of blindness occurs chiefly in neuropathic families, but evidences of nervous disease are rarely associated with it; occasionally, however, patients complain of dull aching pains, or of numbness or paræsthesiæ in their limbs, and in a certain number the knee-jerks and other tendon-jerks are absent.

Pathology.—The symptoms are those of bilateral retrobulbar neuritis, but very little is known of the exact pathology. The disease has been stated to be due to a central retinitis, but of this there is no evidence. Fisher has suggested that the central scotomata may be produced by the pressure of an enlarged pituitary gland on the chiasma, such enlargement being possibly connected with such changes in the sexual glands as occur at puberty and the menopause.

Treatment.—The administration of strychnine and other tonics has been found useful in some cases, but a tendency for the symptoms to regress spontaneously must not be forgotten. Organotherapy with thyroid and pituitary extracts has been tried, but without much success. During the period of development the patient should be kept at rest and on a light diet, and diaphoretics should be tried. Later, when the condition is established, treatment is futile.

GORDON HOLMES.

OPTIC NEURITIS AND NEURO-RETINITIS.—A variety of pathological changes in the optic disc and the adjacent retina are grouped together under the general heading of "optic neuritis and neuro-retinitis." Some of these are true inflammations, to which the term neuritis or neuro-retinitis in its narrower sense may be applied, but in many the principal change is a mechanically produced oedema, the evidences of inflammation found pathologically being only secondary and subsidiary in nature. For these reasons it is advisable to subdivide the subject into groups according to the nature and site of the pathological changes. I propose to deal with it under three main headings:

1. Simple oedema of the papilla (papilloedema).
2. Inflammation of the optic nerve :
 - (a) Involving the papilla (papillitis).
 - (b) Not involving the papilla (retrobulbar neuritis).
3. Primary inflammation or oedema of the retina spreading to and involving the papilla (neuro-retinitis).

1. Under the heading of **papilloedema** comes the large and important group of cases which in the older textbooks was described as optic neuritis. In these cases we find a simple swelling of the optic disc with venous engorgement and sometimes with hæmorrhages (PLATE 23, Fig. 3), accompanied by little or no disturbance of visual function in the earlier stages, though, if unrelieved, leading afterwards to optic atrophy and gradual blindness. Papilloedema is the most important sign of the existence of a tumour or abscess in the intracranial cavity, or of an intracranial disease producing raised intracranial pressure. It is found in 80 per cent. of all cases of brain tumour, and varies in severity according to the site of the tumour. The main factor in its causation is the raised intracranial pressure, which is communicated through the optic foramen to the nerve sheath, with the result that the pressure in the central vein of the retina has to rise in order to maintain the patency of that portion of it which lies in the nerve and crosses the nerve sheath. The lamina cribrosa prevents the raised sheath-pressure from affecting the intra-ocular pressure; consequently, inside the eye we find a raised venous and capillary blood-pressure with a normal tissue-pressure, and we get increased lymph-formation into the tissues adjacent to the lamina cribrosa. At the same time, as the

main, if not the only, channel of drainage of lymph from the retina and optic disc is into the nerve sheath, the increased pressure blocks its escape, so that we have an increased lymph-formation with diminished lymph-drainage resulting in oedema. This oedema shows itself mainly in the looser tissues of the optic papilla, causing them to swell up and project forwards into the eye. It spreads along the optic nerve-fibres into the retina, sometimes for a considerable distance. It obscures the edges of the disc and buries the arteries, but the dilated turgescient veins are usually bulged forwards with the swelling, and the dilated capillaries give rise to pronounced hyperæmia of the disc, so that it becomes indistinguishable in colour from the surrounding retina. The oedema in some cases becomes so great that it lifts up the membrana limitans interna from the nerve-fibre layer, and it may run from the edge of the disc in a wedge-shaped area to the macula, where it settles down into a series of radiating lines of bright white irregular dots and blotches, forming the so-called macular fan. The raised venous pressure gives rise to hæmorrhages; in some cases these are striate in character owing to extravasations in the nerve-fibre layer, or they may be more blotchy and dusky when they develop in the outer molecular layer. Very large hæmorrhages in the nerve-fibre layer may lift the membrana limitans interna; thus they lose their striate character. The nerve-fibres themselves lying in the oedematous fluid begin after a time to swell up and become varicose, and as these varicosities increase in size the fibres disintegrate and form soft whitish areas of degeneration, in which the so-called cytoid bodies are found. Gradually across the lines of greatest strain and distension neuroglial proliferation takes place and secondary inflammatory changes may appear; then the disc begins to take on a whitish opalescent appearance, while the smaller arterioles and capillaries diminish in calibre. Vision rapidly decreases as this opalescence increases, and when this stage is reached there is little hope of restoring function by relief of pressure. The increase of neuroglial tissue is usually steadily progressive, and as it contracts down and as the vessels diminish in calibre the remaining nerve-fibres are gradually more and more atrophied until complete blindness results.

In cases of cerebral tumour, however, the visual function may be interfered with at an earlier stage and in a different method from

OPTIC NEURITIS AND NEURO-RETINITIS

the one above described. Frequently a history of attacks of temporary amblyopia is given; the blindness may be momentary or last for a few minutes, often accompanied by increased headache, giddiness, and sickness. These amblyopic attacks may actually precede the onset of papilloedema, and are most probably due to internal hydrocephalus distending the floor of the third ventricle so that it presses on the chiasma and blocks conduction. In some cases, especially in subtentorial tumours, this pressure, instead of being intermittent, may become constant, and the vision is seriously interfered with. Relief of the intracranial pressure in such cases may give rise to rapid restoration of function, but when the interference with vision is due to cicatricial changes developing in the nerve-head itself the probabilities are against such a restoration.

The ophthalmoscopic changes seen may be summarized briefly in five stages:

(1) Slight blurring of disc edges both above and below with slight venous turgescence and hyperæmia.

(2) Blurring spreads round inner edge and into retina, giving a brush-like appearance to the edge of the swelling. Venous turgescence is more marked, capillaries on surface of disc become distended and visible, but at outer edge of disc the physiological pit may still be seen. Surface of disc elevated above level of retina to the extent of 2-4 D.

(3) Edges of disc completely blurred all round; the physiological pit no longer seen. Arteries buried in the swelling, veins much swollen and tortuous. Striate hæmorrhages in retina round edge of swelling and on down slope. Sometimes œdema spreading towards the macula and forming macular fan. Retina may be thrown into concentric folds, especially on macular side of disc. (Edema running out for considerable distance along main lines of vessels. Soft whitish areas appearing on surface and slopes of the swelling. Elevation 4-6 D above level of retina as measured beyond the macula.

(4) Crest of swelling gradually becomes whiter and opalescent. Disappearance of small dilated capillaries from surface of disc. Edges of disc become faintly visible again. Venous turgescence less, arteries diminished in calibre. Still much swelling.

(5) Swelling subsides. Edges of disc become clear, but considerable disturbance of retinal pigment round it. Colour of disc opaque white. Lamina cribrosa filled in and

solid-looking. Arteries and veins diminished in calibre, and possibly showing whitish lines along their edges. Disc on same level as surrounding retina.

2. Papillitis and retrobulbar neuritis.—In the second group of cases there is a true inflammation of the optic nerve which may affect the intraocular portion and so give rise to papillitis. Ophthalmoscopically, this is not distinguishable from papilloedema except that, as a rule, the swelling is not so intense (3-4 D). The most important differential symptom is the loss of visual function, which may even precede the development of any disc change. It commences usually as a central scotoma, which may spread until it reaches the periphery in all directions. The pupils are dilated and usually show some reaction to strong illumination, but after contracting dilate again at once, even when the illumination is maintained. The appearance of ophthalmoscopic changes in any inflammation of the optic nerve depends on the part of the nerve attacked and on the extent of the inflammation. If the lesion be immediately behind the bulb so as to involve the portion of the nerve in which the central vessels lie, there will be obvious papillitis, but if the anterior part of the nerve is unaffected there may be no obvious disc changes at this time, and only later, after several weeks, will signs of atrophy appear in the disc.

These inflammations may be due to local causes, as orbital cellulitis, posterior scleritis, or infections from the ethmoidal or sphenoidal sinuses. They may also be due to inflammations spreading down the nerve sheath from the intracranial cavity, e.g. tuberculous or syphilitic meningitis. The most important general cause is disseminated sclerosis; often an acute retrobulbar neuritis is the earliest manifestation of this protean disease, and may antedate any of the other symptoms by years. Acute myelitis from other causes, e.g. syphilis or influenza, may be associated with retrobulbar neuritis, and it also may occur after other acute infections.

An important type occurs in families, mostly affecting the males and coming on in early adult life. It is known as Leber's familial atrophy (*see* OPTIC ATROPHY, FAMILIAL). Optic neuritis occurs also in some cases of diabetes, lead poisoning, and arsenical poisoning.

Very considerable restoration of function may follow subsidence of the inflammation, and in some cases quite full visual acuity with normal fields may be regained, though even

then careful examination may show that a small central or paracentral scotoma for green and red has persisted. One definite evidence of a past neuritis is a pallor affecting especially the temporal half of the disc, which may be present even when the visual functions seem to be perfect.

3. Neuro-retinitis.—In this group of cases the optic nerve is affected by disease spreading to it from the retina. As the nerve-fibres spread out and cover the whole of the inner surface of the retina, any retinal inflammation or œdema is liable to pass in towards the disc. It is mainly the central forms of retinitis that are liable to be confused with optic neuritis or papilloœdema, especially albuminuric and syphilitic retinitis. I would, however, include here also the toxic amblyopias, which were formerly regarded as forms of chronic retrobulbar neuritis, but which are much more probably due primarily to retinal poisoning. Tobacco and alcohol are the two principal causes of this form of amblyopia, but other toxins, as carbon disulphide, thine, etc., may produce it, and it also occurs in diabetes. Further, diabetes seems to produce a much greater sensibility of the tissues to the toxin of tobacco.

LESLIE PATON.

OPTIC THALAMUS, LESIONS OF (*see* NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF; Thalamic Syndrome, under PAIN, CENTRAL).

ORAL SEPSIS. A septic mouth has been regarded as a factor in producing or influencing so many general affections that a very long list might be made. Much work has yet to be done to establish clearly a direct relationship between a mouth infection and definite secondary conditions. But it cannot be denied that, from whatever illness a patient may be suffering, the state of the mouth ought never to be disregarded.

Infected teeth have more than a local effect, and give rise to secondary conditions (1) by their influence upon neighbouring parts, (2) by organisms and their toxins being carried to the alimentary tract, and (3) by their being conveyed through the blood and lympho-vascular systems to remote parts.

1. Influence of infected teeth upon neighbouring parts.—Gravity plays an important part in the mouth by causing particles to sink, so that drainage is less perfect from the lower than from the upper part. The sulci, particularly the upper one, are washed by the parotid

secretion, which passes beneath the cheeks and lips by their movements and also by capillary attraction. The sublingual area is bathed by the submaxillary and sublingual saliva as well as that from the parotid, while drainage is aided by the movements of the tongue. *Infection of the sublingual or submaxillary glands* occurs from the mouth, calculus in the duct of the submaxillary gland being not uncommon. *Infection of the parotid gland* takes place when the secretion is diminished; it is important, therefore, to cleanse the mouth carefully in all fevers.

Leucoplakia and the associated superficial changes in the tissues, although often due to a recognized cause such as syphilis, may result entirely from infected teeth, as is shown by the great improvement following their extraction. The commonest site is on the buccal aspect of the third and second mandibular molars. It also occurs along the line of occlusion of the teeth, but over the mandibular teeth rather than those of the maxillæ. A patch occurs at times in the region of the frænum of the tongue. A fissure in the region of the tongue, lip, or elsewhere may be prevented from healing. In the experience of the writer a fissure on the tongue of two years' standing healed almost immediately after local dental treatment, and without extraction of teeth. The removal of dental irritation, particularly of an infective nature, must be of great importance in cases regarded as due to other causes such as syphilis and alcohol.

Friction would appear to play a part in producing these changes, the toxic substance being apparently rubbed into the epithelium. Traumatism may be a factor, for the cheeks are not infrequently bitten when the teeth become worn down. The abrasion produced by the sharp edge of a tooth will heal in a healthy mouth, even though the tooth be retained, but in an infected mouth the ulcer persists and may become the site of an epithelioma.

Pain, occasionally acute, at the side of the base of the tongue may be caused by an infected mouth. There is no abrasion, but a small red patch is seen at this point, and the discomfort may persist in an irregular manner for some time; treatment of the teeth corrects the condition. *Simple ulceration of the mouth* follows an abrasion which becomes infected. The tooth-brush is a common cause, particularly in mouths with a superficial cleanliness which is often misleading. These ulcers are

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described as dyspeptic ulcers, the dyspepsia and ulcer probably being due to the same cause; treatment must be directed to the infected teeth. The ulcers are often very painful, not deep, but prone to extend beneath the epithelium, increasing until they become quite large. Phenol applied carefully to the ulcerated surface relieves the pain.

The gingival form of *Vincent's angina*, though due to specific organisms and contagious, does not occur unless some gingival changes already exist; mouth-breathers are particularly liable to infection. The gums become much swollen, the margins ulcerate and present yellow sloughs on the surface. The presence of the teeth is important, for the ulceration occurs chiefly upon the tooth aspect of the gum; the papillæ between the teeth may be destroyed completely and necessitate extraction. The course is rapid, and extension to neighbouring gums and even to the floor of the mouth and the palate may take place in a few days. The breath grows foul, the patient has fever and is ill, the lymphatic glands become swollen and tender, the teeth loosen and, if treatment is delayed too long, drop out, as the bone supporting them undergoes necrosis. The parts should be swabbed with hydrogen peroxide and a mouth-wash used, which should be retained in the mouth and not ejected immediately. Those liberating chlorine, such as Dakin's solution (1 per cent.), are the most effective. Dental treatment demands the removal of every form of irritant, such as calcareous deposits, overlapping fillings and crowns, treatment of carious teeth, and extraction of teeth if necessary.

Ulcerative stomatitis in children arises in connexion with an infected tooth which is painful, and is therefore avoided. All cleansing processes cease, the opposite side of the mouth is used for eating, and brushing, if practised at all, is restricted to the parts which are insensitive. Débris accumulates upon the affected tooth, most commonly a mandibular temporary molar. The lingual and masticating surfaces are partly cleansed by the tongue, so that the ulceration usually begins on the buccal aspect at the gum margin. It spreads rapidly; a large ulcer is frequently produced on the mucous membrane of the cheek immediately opposite the tooth. The ulceration extends along the gum margins, and the mouth may be extensively involved. The temperature is raised, lymphatic glands are affected, and the child is quite ill. (A growth of strep-

tococci was obtained in a series of cases from which cultivations were taken.) Thorough cleansing of the mouth should be undertaken before extraction of the tooth, unless the pain cannot otherwise be alleviated. Should destruction of tissue have exposed the roots of the teeth, so that they project into the mouth, extraction should not be delayed.

If several teeth need extraction, the chief offender may be removed, the mouth cleansed, and extraction of the remainder carried out after the acute stage has passed. Necrosis may occur if care is not exercised in treatment.

The association of *syphilitic and mercurial ulceration* of the mouth with dental infection has been long recognized, for an endeavour to maintain the cleanliness of the mouth is always enjoined.

Impaction of the "wisdom tooth."—When the third mandibular molar is "impacted," i.e. unerupted, with insufficient space into which to erupt, the most serious changes produced by direct extension from the teeth may supervene. Infection of the soft parts situated above the unerupted tooth occurs and causes ulceration. The patient, who is about 20 to 30 years of age, may complain of "sore throat," and the offending tooth may be unsuspected, as the inflammation spreads to the more lax tissue of the pharynx rather than to the dense tissue situated anteriorly. The whole condition usually subsides, but returns at intervals with the "recurrent sore throat." In due course suppuration, with definite abscess formation, may occur around the tooth and lead frequently to a diagnosis of quinsy, or there may be much swelling of the face and neck in the region of the angle of the jaw. The suppuration usually extends backwards and upwards; when involving the temporal muscle, great difficulty in opening the mouth follows (trismus). This is nearly always due to the inflammatory process, but cases are recorded as due to a nerve reflex. Occasionally months may pass before free movement is completely established, in spite of satisfactory treatment. The more serious conditions urgently need surgical interference, for suppuration may extend into the neck. If external to the cervical fascia, it has been known to reach the clavicle; if internal to the fascia, Ludwig's angina results, with great swelling in the suprahyoid region, whilst œdema of the larynx may prove fatal. The toxæmia is severe, and the patient is seriously ill. Septic phlebitis of the pterygoid plexus of veins may lead to

cavernous-sinus thrombosis, and cerebral abscess also has supervened.

Drainage of the area deep to the temporal muscle should be attempted in cases which present great difficulty. Removal of the tooth is necessary in recurrent cases; extraction of the upper third molar may relieve the cause of irritation and give time. Extraction of the mandibular second molar is advocated, and may be imperative, but it usually inflicts a serious loss on the patient, for the third molar rarely comes into good position, even should its removal be rendered unnecessary.

In slight cases an incision corresponding to the outer aspect of the tooth may give permanent relief; the cheek should be rendered taut by a finger of the left hand, the incision being made parallel to its inner side. Lini-mentum iodi, if applied carefully, may palliate; in most cases extraction of the maxillary molar is the simplest and most certain form of alleviation. Constant irrigation is most important; the socket tends to collect debris, and unless it is filled with bloodclot drainage is very imperfect. Simple cases, if neglected, often give rise to severe pain due to an osteitis which is very persistent and does not readily yield to treatment. A saturated solution of chloretone ($\frac{1}{2}$ gr. to 1 oz.) in water is valuable for syringing, or it may be held in the mouth alternately with eusol or some similar lotion.

An ulcer occurring in edentulous patients or in a part of the mouth where teeth have been lost is nearly always due to a *badly fitting denture*. It is most commonly seen in the sulcus between the lip and the gum. For the sake of appearance the denture is worn in spite of ulceration, and with continual use large masses of granulation are produced, over which epithelium may grow in part, giving rise to a condition which may be mistaken even for a malignant growth.

A denture which has been worn for some time may cause ulceration of the palate; this is usually due to absorption of the alveolar ridges, permitting the pressure to come upon the palate, which undergoes little change. Treatment of these conditions demands removal of the denture and the frequent use of mouth-washes, a correctly fitting denture being made later.

Necrosis of the jaw.—The commonest forms of jaw necrosis are directly associated with the teeth, either (1) in the region of the apex when infection has reached the bone from the pulp,

or (2) at the margin of the socket in cases of pyorrhœa alveolaris. (1) *Infection of the bone from the pulp* leads to an acute alveolar abscess (see ABSCESS, ALVEOLAR). If the tooth remains and is not treated, or the pulp canal is not rendered sterile, a *dental sinus* results. The dead bone may be absorbed, and the sinus become lined with granulation tissue, but with a constant discharge the area of destruction in the region of the apex of the tooth may gradually increase, though this rarely happens. (2) The *marginal necrosis* arising in cases of pyorrhœa is considered in the article on PYORRHOEA ALVEOLARIS.

Exposure of the roots of the teeth as the result of *violent brushing* (formerly called "erosion") leads to infection of the bone, particularly between the roots of the molars, where granulation tissue may be present, and bare bone be quite commonly found also. Destruction may be so considerable that a dental probe can be passed completely beneath the tooth between the roots, pyorrhœa alveolaris having been produced artificially.

Rarefaction of bone or the formation of small sequestra occurs as the result of *dental operations*. The use of arsenious anhydride to destroy the pulp may cause a marginal necrosis which continues for a long period unless treated. This condition may be produced also by other drugs, but is unusual.

Infection of the bone may follow injury by dental instruments; perforation of the root, either through the side or the apex, is the commonest. Instruments may be broken and left in the tissues, such as a needle used for an injection, or a bristle employed for removing the pulp.

Necrosis following tooth extraction occurs more often in the mandible than in the maxillæ, most frequently when the bone is injured at the operation. Acute necrosis involving a large part of the jaw, more frequently the mandible, may arise, especially in children, from infection by a tooth. It is associated with debility and occurs in specific fevers (*exanthematous necrosis*). In children it may be accompanied by ulcerative stomatitis.

In all cases of extensive necrosis its course indicates a phlebitis of the subalveolar (central) vein. Drainage must be established and frequent irrigation employed, but great care must be exercised to avoid removing the sequestrum until it is loose, or deficiency in the bone may result.

Phosphorus necrosis is due to infected teeth.

ORAL SEPSIS

a fact which has been so well recognized that dental treatment and inspection are compulsory in match factories.

Bad dental work is the cause of infection becoming established in many ways. Ill-considered extraction of teeth, particularly of the first molars, causes the neighbouring teeth to lean towards the gap. The surface directed towards the gum is difficult to keep clean, food débris collects in the angle, and a pocket nearly always results with serious consequence to the tooth; also by spacing of the teeth food may be forced between them, causing injury to the gums and subsequent infection. Spacing of the molars and premolars may allow the jaws to close nearer together, so that the lower incisors, impinging upon the upper ones, tend to force them outwards and cause them to protrude and become spaced. This condition is more pronounced in patients who have lost several of their molars and premolars. The power of mastication may be considerably reduced, as the remaining teeth meet at points and the usual crushing surfaces do not meet. Collar crowns, bridges fitted to rest upon the gums, and fillings inserted with overlapping edges at the gum margin must be regarded as serious sources of infection.

The treatment of root canals is so rarely carried out aseptically that "dead teeth" are regarded by many as always infected. It is possible, however, to carry out the operation satisfactorily.

Infection of the naso-pharynx from the mouth occurs by direct extension, and without treatment of the mouth little benefit accrues from the treatment of the naso-pharynx. In a remarkable series of cases the writer was able to effect the cessation of chronic otorrhœa in children by the removal of infected temporary teeth; the usual treatment of the ear had been carried out for a considerable period, but with little benefit until the mouth was rendered healthy.

Sore throat and quinsy due to infection associated with impacted wisdom teeth is frequently overlooked.

Few surgeons will undertake the operation for removal of tonsils and adenoids until the mouth has been rendered healthy. Though more remote, *septic pneumonia*, attributed to the administration of an anæsthetic, is in almost all cases due to infection from the mouth. Gross infection by the inhalation of particles occurs very readily, particularly when the mouth is forcibly opened.

2. Influence of oral sepsis on the alimentary tract.—The alimentary tract may become affected by micro-organisms or their products conveyed from the mouth to the stomach, and in some cases to the bowel beyond. The power of the stomach to destroy such organisms must be exceedingly great, but when this resistance breaks down, the difficulty of restoring it is often remarkable. In gastric dilatation an infected mouth may prevent any benefit following treatment. Briefly, removal of infection from the mouth is imperative in all gastric or intestinal disorders; it is unnecessary to enumerate all the pathological changes which might be influenced by an infected mouth. Some cases of mucous colitis, appendicitis, pancreatitis, cholecystitis, and other diseases have been definitely attributed to it. Dyspepsia is generally recognized as being associated with it, and is sometimes attributed to a deficiency in masticatory power apart from infection, yet, if the latter is removed without restoration of the former, great improvement often results. This, however, is not to depreciate the importance of mastication.

3. Influence of oral sepsis through the blood and lymph. Knowledge of the influence exercised by micro-organisms or their toxins through the blood and lymph-vascular system is largely limited to clinical experience. Identical organisms have been found present in the mouth and in a remote area, suggesting a definite relationship, but much work has to be done to place our knowledge on a more scientific basis. In the writer's experience, dental treatment has resulted in benefit or complete relief in a great variety of cases. In some the association between the secondary site and the teeth cannot be explained, but in a large number an infected mouth has led to changes in a part which has been injured, such as a joint, or a part which has had excessive use, such as the hand of a pianist. It would appear that either organisms from the mouth may become located elsewhere, or the tissues may be affected by the toxic products alone; in some cases the rapidity with which a secondary site clears when the mouth is freed from infection suggests the latter relationship. Perhaps the commonest of the general disorders which may have their origin in an infected mouth are the large group formerly included under *rheumatism*. The change is usually of a chronic character, but occasionally may be subacute or acute. The form affecting joints may be regarded as most

favourable for treatment when the condition is periarticular, or, if articular, when acute or subacute. The dental treatment of rheumatoid arthritis, except in the early stages, is often disappointing. It would seem that a definite secondary infection results, and that though dental treatment may lead to temporary improvement by lessening the general infection, it is often insufficient to effect a cure.

Apart from joints, osteitis may follow mouth infection. Muscular and articular pains, both localized and vague, are often due to dental infection, as also are lumbago, sciatica, and the changes known as fibrositis. These "rheumatic" patients, particularly in cases of long standing, often have characteristic pallor and a smooth, shiny skin, most obvious in the forehead and hands.

In *neurasthenia* the mouth should be examined most carefully, for some effect upon the central nervous system is present in nearly all patients suffering from toxic absorption from the mouth. The patient becomes more easily tired, often irritable, does not sleep so well, and is sometimes very restless. In two patients, who recovered after dental extraction, the state of restlessness was so extreme that they were not allowed to be left alone, the mental balance being definitely disturbed. Treatment of the teeth has resulted, too, in complete cure of cases of *exophthalmic goitre*. *Eye infections* and changes due to toxic absorption may originate in dental infection; occasionally recovery is quite dramatic in these cases. Although *anæmia* occurs in a great many cases, dental treatment as a possible means of effecting a cure in the more serious forms is undertaken with less confidence than formerly. The *cardio-vascular system* may be affected by a toxæmia of dental origin, as in the case of an elderly man who suffered from giddiness which was almost entirely relieved as a result of dental treatment.

The relationship between an infected mouth and a "rise" of temperature is important. Acute conditions, as alveolar abscess, cause fever, but such a change is quite uncommon with chronic infections. When, exceptionally, a rise occurs it may, however, be misinterpreted.

Dental infection provides the commonest cause of *enlargement of the lymphatic glands in the neck*, and the improvement that follows dental treatment is remarkable. The enlargement of glands anterior to those caused by unhealthy tonsils is usually of dental origin. They are sometimes mistaken for tuberculous glands.

Treatment of oral sepsis.—The mouth must be rendered healthy. This will rest mainly with the dentist, but a great deal can be done by the medical practitioner. The tooth-brush must be used with care; it should have stiff bristles and be quite small to allow access to every part of the mouth. Two movements should be practised. One is across the teeth commencing upon the biting surfaces, then upon the lingual and buccal aspects; the movement should be short, brushing being directed to one tooth at a time; the points of the bristles should be used by means of firm pressure, but not sufficient to bend or break them down. The other movement is downward upon the maxillary and upward upon the mandibular teeth. The side of the brush is placed on the gum and a sweeping movement made, turning it as it passes over the gum and teeth. If pain is caused the movement is wrong. While the mouth is infected a lotion of citric acid, 5 gr. to the ounce of water, with 1 min. of formalin to preserve the lotion, is more serviceable than tooth-pastes. Vinegar or weak acetic acid and water will be found convenient for hospital patients. The brush should be washed each time before being dipped into the lotion. Tooth-powder is dangerous, because particles of it are apt to lodge in the pockets. Particles of chalk are more readily removed from the mouth when soap is used; it is an ingredient of most tooth-pastes. The tooth-brush should be washed most carefully; if the mouth is badly infected, it should be soaked in a weak formalin solution each time after use. Butter muslin with a wide mesh is valuable for rubbing the gums, particularly if the mouth is too sore for a tooth-brush to be used; pressure applied gently but with increasing firmness may be tolerable when rubbing is too painful: food particles are forced from beneath the gum, and the friction removes the superficial epithelial cells, but perhaps the most valuable effect is that derived from emptying the chronically congested blood-vessels. The gauze may be soaked in hydrogen peroxide 10 vols., one part to three parts of water.

A mouth-wash used for about ten minutes at a time at frequent intervals is indicated. Eusol is most valuable in badly infected mouths, whilst a hot carbolic wash (1 in 120) is most comforting when the mouth is painful; additional relief may be obtained by using chloroform water with phenol.

WARWICK JAMES.

ORBIT, AFFECTIONS OF

ORBIT, AFFECTIONS OF.—Pathological conditions of the orbit manifest themselves clinically by enophthalmos or by exophthalmos (proptosis).

Enophthalmos is rare, and is usually associated with a traumatic history, an injury of the orbital wall permitting of a dislocation of the orbital contents towards a neighbouring air sinus. In some cases it is due to a cicatrizing process, the result of a diffuse orbital cellulitis. It is sometimes present in paralysis of the cervical sympathetic, and occasionally after an advancement operation for strabismus.

Exophthalmos may be bilateral or unilateral. The prominence of the globes varies so much in different individuals that in bilateral cases one may be confirmed in the suspicion of a pathological exophthalmos only by the history of increasing proptosis and by other signs of disease.

Test for unilateral exophthalmos.—Typical cases are obvious, and the prominence of one cornea can be detected easily. In slight cases considerable care in examination has to be taken. The best test is that of examining the relative positions of the cornea from a point above the centre of the forehead; the patient is seated and looks straight in front or somewhat downwards, while the surgeon, using one eye only, bends forward over the patient's head, lifting the upper lids with the forefingers as may be necessary.

Having made a diagnosis of exophthalmos, certain further points should be noted:

(1) The direction of displacement of the globe, whether directly forwards, or in other directions, e.g. downwards, as well.

(2) The presence or absence of limitation of ocular movements on the affected side.

(3) Any inflammatory condition of the lids or conjunctiva, e.g. oedema or chemosis.

(4) Any palpable swelling coming forward between the bone and the globe, and its degree of solidity or fluidity.

(5) The possibility of reducing the proptosis by steady pressure, or whether the resistance is very firm.

(6) Whether there is pulsation on pressure of the globe backwards.

Bilateral exophthalmos (1) may be a symptom of Graves's disease, in which case other ocular signs and other symptoms of that affection will be present (see EXOPHTHALMIC GOITRE); or (2) may be due to extensions of sphenoidal sinus growths or inflammation, or may occur

in the later stages of cavernous-sinus thrombosis (see below).

Unilateral exophthalmos is due to the following conditions:—

(a) **Orbital cellulitis**, acute or chronic. **Etiology.**—The acute forms are met with in adults as an extension of inflammation from the nasal sinuses, especially the ethmoidal cells; in children the condition sometimes occurs as the result of an acute periostitis of the bony wall, similar to the acute osteomyelitis of the long bones. Other causes are metastatic abscess, extension of sepsis from the teeth, the lachrymal sac or gland, facial erysipelas, and suppurating wounds, with or without retention of a foreign body.

The chronic cases also may arise from the nasal sinuses, including the frontal sinus, or from a gummatous or tuberculous periostitis.

Symptoms.—The disease in the acute cases is associated with moderate pyrexia, pain, diminution of vision, impaired movement of the globe, proptosis and displacement of the eyeball away from the initial focus of the inflammation; the lids early show some oedema, and the conjunctiva may become chemosed. The fundus oculi usually exhibits no change. In chronic cases pyrexia is absent, and the signs of oedema are less evident or not present at all.

Diagnosis.—The distinction from thrombosis of the cavernous sinus may be difficult, but is important. In thrombosis there are early immobility of the globe and interference with the patient's mentality, and later, oedema of the lids and the other orbit; there may be signs of interference with the circulation in some of the emissary veins of the skull. In acute panophthalmitis there is oedema of the orbit as a result of intra-ocular suppuration. The presence of the latter will be evidenced by iritis, hypopyon, pus behind the lens or in the pupil, etc. To distinguish orbital cellulitis from tumour of the orbit, in chronic cases, is sometimes difficult. A rhinological examination is necessary, and a skiagram may be useful. Inflammatory cases on the whole tend to interfere with the ocular movements more than does a new growth, and the position of the swelling may be suggestive, e.g. a globe may be pushed forwards and outwards by a collection of pus spreading outwards from the ethmoidal cells.

Prognosis.—The acute cases are serious; infection may spread backwards to the cavernous sinus, or through the roof of the orbit

to the frontal lobe or meninges. Most cases finally do well, and often recover useful vision.

Treatment.—In the early stage, in acute cases, general measures against inflammation must be taken, e.g. rest in bed, regulation of the bowels, hot fomentations to the eye, etc. A successful search for the cause will indicate specific treatment, e.g. the opening-up of infected ethmoidal cells. If pus has evidently formed, early incision must be undertaken to prevent the complications just mentioned, but an idea of its position may sometimes be obtained by waiting a day or two for the abscess to come forward. If, however, it is far back, more than one incision may be necessary before it is located, and must not be too long delayed.

In chronic cases operation is generally necessary; even a broken-down gumma may have to be incised, under aseptic precautions, in order to relieve pressure on the optic nerve.

The incision should be free, through the skin down to the orbital margin, whence the orbital tissues may be separated, keeping close to the bone. Damage to the pulley of the superior oblique, the lachrymal sac and gland, must be avoided.

(b) **Septic thrombosis of the cavernous sinus** is invariably fatal. It is caused by spread of infection to the cavernous sinus from the middle ear, and from septic conditions of the face and throat. Its main symptoms are described in connexion with the diagnosis of orbital cellulitis. (See also CEREBRAL SINUSES, THROMBOSIS OF.)

(c) **Orbital tumours** are uncommon. Endotheliomata, sarcomata, and fibromatosis of the optic nerve occur and displace the eyeball directly forwards. Other tumours may arise outside the cone of the recti muscles, especially from the periosteum, e.g. endotheliomata or osteomata, and these may displace the globe laterally as well as forwards. As a rule, the ocular movements are not much interfered with, as the muscles are not infiltrated. The diagnosis is often difficult. Treatment may necessitate exenteration of the orbital contents, but in some cases it is possible to remove a benign tumour without excising the eyeball.

(d) **Pulsating exophthalmos** is usually due to aneurysm of the internal carotid artery, or an arterio-venous anastomosis between the artery and the cavernous sinus. There is exophthalmos with pronounced pulsation, the patient complains of buzzing noises in the head, and with a stethoscope a bruit can be

heard in the orbit. The condition is often associated with a history of trauma, and may necessitate ligation of the carotid artery, which is sometimes attended with success.

(e) **Surgical emphysema** is an occasional cause of proptosis, associated with fracture of the outer wall of the nose. Air-bubbles may be seen under the conjunctiva. The treatment consists simply in not blowing the nose for some days.

(f) **Orbital hæmorrhage** is most often met with as the result of gunshot wounds, but may also occur from fractures of the base of the skull, involving the anterior fossa, and from abnormal blood states, e.g. scurvy.

(g) In **paralysis of the recti muscles** the globe may come forward as the result of the loss of muscular tone. This is occasionally seen after a free tenotomy for strabismus.

F. A. JULER.

ORCHITIS.—Inflammation of the body of the testicle. In epididymitis the inflammation extends, as a rule, from the epididymis to the body of the testis; but in orchitis, though the epididymis may occasionally be affected to a certain extent, the body of the testis is the structure essentially and primarily involved.

Etiology.—Acute orchitis may occur in the course of certain fevers, especially mumps, but also in typhoid, smallpox, scarlet fever, and influenza, and is a rare complication of rheumatism, malaria, and tonsillitis. It may also be caused by injury, and is occasionally due to gout. In traumatic orchitis, which may be accompanied by epididymitis, the primary condition is probably a contusion, upon which the inflammation follows. A true gouty orchitis may occur, either during a typical attack, or in a gouty subject apart from any affection of the joints: it must be distinguished from epididymo-orchitis secondary to gouty urethritis.

Course.—When secondary to one of the fevers, infection takes place by the bloodstream. Typhoid orchitis may terminate in suppuration, but this is rare in other forms, and does not occur with mumps. There may be some effusion into the tunica vaginalis, but not enough to mask the disease. Orchitis is not an uncommon complication of mumps, its frequency varying in different epidemics. It generally terminates in resolution, and the testicle suffers no permanent harm. Atrophy, however, occasionally occurs, whatever the cause of the orchitis. This serious termination is not uncommon with the orchitis of

mumps. In some epidemics it is stated that as many as one-third of the cases of orchitis have terminated in this way.

Symptomatology.—The body of the testicle enlarges rapidly, forming a painful, tender swelling. The pain varies from a continual dull aching to acute pain radiating along the cord to the groin and back. The scrotum may be red and œdematous, and there may be a small hydrocele. There is generally some pyrexia, and in the more acute cases there is severe constitutional disturbance. In traumatic orchitis the pain, which is very severe and is generally described as "sickening," immediately follows the injury, as also does the swelling.

When orchitis is due to mumps, boys and adolescents are usually attacked, young children and older patients not often being affected. The orchitis generally develops about the eighth day, when the parotid swelling is subsiding, and is often preceded by a rise of temperature. The swelling of the salivary glands may be trifling, or may even be absent, or may appear after the orchitis. The other testicle may be affected as the first improves.

In typhoid fever, orchitis may occur either in the course of the disease or during convalescence. It is generally not severe, and, though it takes a long time to clear up, usually terminates in resolution. Suppuration may, however, occur with disorganization of the testicle. Orchitis is a rare complication of smallpox, influenza, and scarlet fever. In scarlet fever suppuration has been recorded as an occasional termination.

Pain, swelling, and œdema which persist, or increase, in spite of treatment, suggest the presence of pus, and an area of softening indicates the situation of the abscess.

Diagnosis.—The diagnosis of orchitis does not usually present much difficulty. Care must be taken to distinguish it from *acute epididymitis* secondary to infection of the prostatic urethra.

Treatment.—In any case of orchitis the patient must remain in bed. The scrotum should be kept well raised, and hot lead and opium fomentations applied. A saline purge should be given, and small doses of pulv. ipecac. co. are of use in relieving the pain. In the most acute cases, for instance after injury, injections of morphia may be required. A suspensory bandage should be worn for some time after the acute symptoms have subsided. Attention must also be directed to the disease

of which the orchitis is a complication. For instance, in malaria quinine should be given, and where gout or rheumatism is suspected as the cause, treatment appropriate to those diseases should be carried out. When suppuration occurs, incision and drainage will be required, and in some cases where disorganization and sloughing of the testicle have taken place orchidectomy will be necessary.

PHILIP TURNER.

ORGANOTHERAPY.—A method of treatment based on the physiological conception of the internal secretion of specific stimulant substances by certain organs. The substance or extracts of such organs may be administered to compensate for a defect of natural activity, or to produce a local or temporary exaggeration of the specific action. The rational use of the method is clearly restricted to organs which furnish internal secretions, and with these it is only likely to succeed when the active constituent is a fairly stable substance, and is stored to some extent in the gland which forms it. The fulfilment of these conditions in the case of the thyroid gland probably accounts for the efficacy of its administration—the first, and still incomparably the most important and successful, application of the method.

The thyroid gland.—The earliest success in thyroid medication was obtained with a glycerin extract of the gland, derived from sheep or oxen. This has almost entirely been replaced by the desiccated gland-substance, which is given by the mouth in compressed tablets, or as a powder in cachets. There is some evidence connecting the activity of the thyroid substance with the iodine which the gland contains in a much larger proportion than any other tissue of the higher animals. It has been suggested, therefore, that the dried gland-substance should be standardized for iodine content, and, until some better standard is available, it is advisable to use material so standardized. It is customary to express the dose in terms of the weight of fresh, moist gland-substance represented.

During the last few years Kendall has described the isolation in crystalline form of a substance which appears to have a good claim to be regarded as the pure active principle of the thyroid gland. From such details as are available it is an iodine-containing indol derivative. If this substance becomes obtainable in quantity it will probably supersede

the crude gland, and render thyroid medication much more exact.

Administration of thyroid substance or extract is a specific treatment for all conditions due to deficient action of the gland. Well-marked cretinism and myxœdema are so well characterized that probably few cases now escape recognition and treatment in civilized communities. In myxœdema some advocate the administration, at the commencement of the treatment, of the maximum dose which the patient will tolerate without showing symptoms of thyroid excess. Such authorities give up to the equivalent of 15 gr. of fresh gland per diem in severe cases. Others proceed more cautiously, giving, perhaps, 5 gr. three times a week, and increasing gradually the frequency and size of the dose until the patient is taking as large a daily quantity as can be borne without producing cardiac distress and myalgia. Loss of weight will, of course, always occur if the treatment is efficient, since the essential action of the thyroid principle is to accelerate metabolic breakdown. The aim in each individual case should be so to adjust administration that the patient does not accumulate the waste material at such a rate as to produce fever, cardiac distress, or other untoward symptoms. When once the normal condition has been regained, the dosage is reduced until the minimum quantity is found which suffices to maintain the bodily functions and to prevent any tendency to relapse. In most cases such reduced dosage must be continued throughout life, the treatment being essentially substitutive and not curative. Cases are on record, however, in which an inadequately functioning gland seems to have recovered during the rest afforded by a course of thyroid treatment, and in which it has, accordingly, been possible later to discontinue the administration altogether. Children are said to bear thyroid treatment well, and the young cretin can usually take from one to five grain-doses daily, according to age. The urgency of beginning treatment early is obvious.

Many conditions have been attributed during recent years to minor or partial thyroid deficiency, but it is beyond the scope of this article to describe them in detail. In the young, slow growth, delayed puberty, defective dentition, and weak hair-growth may be mentioned. Enuresis also may specially be noted, as yielding in many cases to treatment with small doses of thyroid. In the adult almost the whole of the symptom-complex referred

by some observers to intestinal stasis or auto-intoxication—lassitude and depression, sallow, pigmented skin, low body-temperature, joint-pains, headaches, as well as the constipation itself—is attributed by others, from a different point of view, to thyroid inadequacy. Menstrual disorders, either of excess or of suppression, an otherwise unexplained tendency to abortion, and uterine infantilism are similarly laid to the account of a defective thyroid action. In all such conditions, the test of the connexion with lack of thyroid secretion is the efficacy of administering thyroid substance. Large doses should not be needed, and if a daily administration of the equivalent of 5 gr. is not soon followed by signs of improvement, the suggestion of thyroid inadequacy may probably be rejected. Naturally, it is in these cases of minor or relative thyroid defect that there is most hope of being able to discontinue the treatment gradually when the normal condition has been regained.

The possibility should not be left out of sight that thyroid administration may do good in some conditions which are not necessarily due to defect of the natural secretion. Obesity is often associated with other signs of thyroid deficiency, but it is not necessary to assume that every case of obesity, in the absence of such other signs, is due to inadequate thyroid activity. Thyroid administration accelerates the metabolism of proteins and fats in the normal subject, and may therefore be a valuable aid to dieting in the treatment of obesity, even when the natural thyroid function is adequate to most of the bodily needs.

The parathyroids.—Complete extirpation of the parathyroids causes fatal tetany in animals. This has been stated to be accompanied by excessive excretion of calcium, of which the tissues become relatively depleted. More recent evidence connects it with an abnormal metabolism leading to the formation of methyl-guanidine, injection of which causes similar symptoms in the normal animal. Some evidence has been produced connecting certain cases of tetany in man with degeneration of the parathyroids. In no such case does administration of parathyroid substance appear to have been beneficial. An attempt has been made, again, to connect paralysis agitans with parathyroid defect, though the view has no good pathological support. Administration of parathyroid substance, or of a nucleo-protein therefrom, has been found by some to alleviate

the condition, while the experience of others has been entirely negative.

For clear evidence of the possibility of supplementing deficient parathyroid secretion by administering the gland-substance, we are dependent hitherto on reports of a few cases. In one remarkable case, after operation for removal of a goitre, a condition arose resembling Graves's disease in many features, but with no exophthalmos. Administration of thyroid substance made this patient worse, but his condition improved rapidly when small doses of dried parathyroid were given.

It must be borne in mind that the sheep's thyroid, ordinarily used in thyroid treatment, contains the substance of the internal pair of parathyroids. It is possible that some of the reputed actions of the thyroid preparations, e.g. the beneficial action which has been ascribed to injection of an extract in eclampsia, ought to be attributed to the included parathyroid substance.

Pure parathyroid substance, for whatever purpose it is given, is administered in small doses, such as $\frac{1}{10}$ gr. of dried parathyroid, several times a day.

The suprarenal capsules.—The relation of Addison's disease to suprarenal defect, usually as the result of tuberculosis, is perfectly clear. Not so clear is the share in its causation taken by defect of the internal secretion of the cortex and that of the medulla of the gland respectively; probably both are concerned. Unfortunately, there is relatively little to be hoped from substitutive therapy. According to Grünbaum a rise of blood-pressure, following the oral administration of the gland-substance, is diagnostic of Addison's disease. The administration of large doses, 20-30 gr. thrice daily, is said to have some effect on the course of the disease; but a delay of the inevitably fatal issue is the most that can be hoped for.

The therapeutic importance of the suprarenal glands depends almost entirely on the presence in their medulla of the acutely acting hormone adrenalin. This is the only hormone of which the chemical constitution is yet definitely known. Its structure has been elucidated, and it can now be prepared synthetically. Largely through the work of the Cambridge School, and especially of Elliott, it has been found that this principle, adrenalin, reproduces in its action, with great precision, the effects of stimulating the nerves of the true sympathetic system.

To a minor extent adrenalin is administered

to supplement a depleted natural supply in conditions of exhaustion following acute fevers, and particularly diphtheria. The object being a slow replenishment, and persistent mild tonic action, adrenalin is given in such cases by the mouth or the rectum. A dose of 1 mg. (1 c.c. or 15 to 20 min. of 1-in-1,000 solution) may so be given several times daily without harm. In shock following surgical operations, again, adrenalin has been used, 20 min. of 1-in-1,000 solution, diluted with a few pints of warm saline, being slowly infused into a vein. The most recent observation has tended to throw doubt on the value of this use of adrenalin.

The extraordinarily rapid and potent, and no less remarkably evanescent, effects of very small doses of adrenalin are only seen with intravenous injection. So given, it produces a faithful picture of sympathetic stimulation. General arterial constriction and increased activity of the heart drive the blood-pressure rapidly to a remarkable height; other plain muscle receiving a motor sympathetic supply, such as that of the uterine wall, is thrown into powerful tonus. On the other hand, the plain muscle of the bronchioles and the alimentary tract, being inhibited by sympathetic nerve-impulses, loses tone and rhythm in response to adrenalin. If adrenalin, even in small quantity, gets quickly into the blood-stream, the rise of the blood-pressure suddenly from the normal level to a great height may, in any case, produce an alarming reflex inhibition of the heart through the vagus. Under chloroform anaesthesia, especially if light, there is a serious danger of the sensitive heart being thrown into fatal fibrillation of the ventricle, if adrenalin gets too quickly into the circulation. Injection of adrenalin into a vein, or even into the vascular mucous membrane of the nose or the mouth, under light chloroform anaesthesia is, therefore, a very dangerous procedure. This is especially worthy of emphasis, in that the commonest of all the uses of adrenalin is to produce a local ischaemia of mucous membranes or subcutaneous tissues, by causing constriction of the arterioles in the area to which it is applied, or into which it is injected. It is thus used by simple superficial application, in removing the hyperaemia of an inflamed conjunctiva, or in producing a bloodless field of operation in ophthalmic or nasal surgery. A 1-in-1,000 solution of the hydrochloride, or borate, is commonly employed for this purpose. It is frequently used in combination with a

local anæsthetic, the action of which it favours by restricting absorption; since it thereby concentrates the anæsthetic action in the neighbourhood of the injection, and diminishes the danger of a general toxic effect. For injection under the skin or mucous membrane with local anæsthetics, adrenalin solutions of 1-in-10,000 to 1-in-100,000 strength are commonly employed. Its local constricting effect on arterioles is of great value in checking bleeding from small abrasions, etc. Thus the 1-in-1,000 solution is applied to the nasal mucosa in epistaxis, or to a bleeding tooth-socket after extraction. Weaker solutions are used to irrigate the bladder, in order to stop bleeding from the mucous membrane. In bleeding gastric ulcer, adrenalin is given by the mouth in small doses at frequent intervals; but this is essentially a local use of the substance, absorption from the alimentary canal being slight and slow. To bleeding piles the free base is applied in an ointment. In hay fever a solution of one of the salts is used in a spray to the nasal and conjunctival mucous membrane, or the free base can be used as a constituent of a snuff.

Of the general effects after absorption, apart from those already mentioned, that on the bronchioles appears to be of the most practical importance. A small hypodermic injection of the 1-in-1,000 solution (1 to 3 minims) acts like a charm in many attacks of asthma.

Injections of adrenalin have been stated to have a beneficial action in osteomalacia. The mode of action is not clear.

The pituitary body.—Like the suprarenal capsules, the pituitary body consists of two main lobes. Between these is an epithelial pars intermedia. The larger anterior lobe has an important influence on growth and development. The smaller posterior, infundibular, or nervous lobe contains, like the suprarenal medulla, a powerful and acutely acting hormone, or possibly a series of such hormones. Defect of the internal secretion of the pituitary body is associated with the condition of obese infantilism described by Fröhlich. The action of both lobes would appear to be deficient in this condition, anterior-lobe defect accounting for the retarded growth and genital infantilism, while the obesity is associated with a greatly increased carbohydrate tolerance, which Cushing associates with deficiency of the posterior lobe. A febrile reaction, following injection of anterior-lobe extract, is diagnostic of deficiency of that lobe, according to the same authority.

Some success in the treatment of this condition of pituitary deficiency has been obtained by administering the substance of the whole gland by the mouth. The success is not of the same order as that resulting from thyroid administration in the analogous deficiency. Relatively very large doses of the pituitary gland are required, and, in many cases, enormous amounts (up to 300 grm. daily) must be given in order to obtain definite improvement. Considerations of quantity and expense may have stood in the way of a general success of the treatment. At the same time, there should be no great difficulty in obtaining adequate quantities of anterior lobe for treatment of cases in which this lobe is solely or mainly deficient, since large quantities of the glands are now used in preparing the widely used posterior-lobe extract.

The extract of the posterior lobe, when injected intravenously, produces powerful and characteristic physiological effects, which may be roughly summarized as a stimulation of plain muscle, and accelerated secretion of urine and milk. Apart from its direct stimulation of plain muscle, it appears to render that tissue more responsive to the excitatory effect of adrenalin. It is stated that contraction of the uterus is caused by a different principle from that which produces rise of blood-pressure, and the promotion of the urinary flow has similarly been attributed to a separate principle. From the point of view of practical therapeutics, however, the matter of chief importance is that a simple watery decoction of the perfectly fresh infundibular lobe possesses these various activities in very uniform degrees. It is usual to employ an extract made with 10 parts of water to 1 of the fresh substance; this is given by intramuscular or intravenous injection, 1 c.c. being an average dose for an adult, though larger doses may be given apparently without danger. It is important to remember that one dose produces a condition of tolerance of further doses, lasting for some hours. One full injection is, therefore, much to be preferred to small doses at intervals.

Though the extract of infundibulum accelerates the secretion of urine in the normal animal, impaired activity of this lobe is associated with a pathological increase of the secretion—diabetes insipidus. A number of cases are now on record in which a daily dose of the extract (1 c.c. hypodermically) effectively checked the excessive diuresis. There is no record of cure.

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The chief therapeutic effects for which the extract is thus used are as follows:—

1. To restore tone to the arterial system, thus indirectly improving the heart-beat and, by the combined action, producing a sustained rise of blood-pressure in surgical shock or collapse. For threatened heart failure in acute fevers the extract has been similarly used.

2. To restore tone and peristaltic activity to the paretic bowel after abdominal operations.

3. To stimulate uterine activity in a labour protracted by uterine inertia, and to promote contraction post partum. If this potent stimulant is to be used during labour, it is of the greatest importance to exclude mechanical obstruction. Several cases of rupture of the uterus through its employment are on record.

4. To check the excessive diuresis of diabetes insipidus.

5. As a galactagogue it has prompt action, but the ultimate benefit is doubtful. In a normally lactating animal the accelerated secretion following an injection seems to be compensated by a following period of diminished activity. Where the supply is naturally deficient it should be given a trial.

Like adrenalin, the posterior-lobe extract has been credited with remedial action in osteomalacia. It is also said to have produced good effects in Graves's disease. These uses have, as yet, no clear rational basis.

The gonads.—Treatment of senility with testicular extract has some interest, in that the first attempt at organotherapy took this form. The results were unconvincing, and similar attempts, continued to the present day, to treat various conditions by injecting or feeding with testicular extracts or substance have failed to produce any decisive evidence of the value of the method.

Ovarian substance is commonly administered for the relief of the nervous troubles attendant on the menopause, whether natural or induced by oöphorectomy. In amenorrhœa also its administration has been recommended, and it has been suggested that the luteal tissue is the constituent that is of value in this direction. On the other hand, the corpus luteum has been supposed by others to be concerned in the suppression of the menses during pregnancy, and the administration of ovarian substance containing luteal tissue has accordingly had considerable vogue in the treatment of threatened abortion. The two lines of treatment seem to involve contradictory assumptions; and, indeed, the value of adminis-

tering ovarian substances in any case is not supported by much stronger evidence than that of the analogous treatment with testicular substance.

The thymus.—The thymus consists chiefly of lymphoid tissue, and there is no evidence that it forms an internal secretion. It occasionally contains nodules of accessory thyroid or parathyroid tissue, and the presence of these may possibly account for some of the effects which have been alleged to result from its administration. It has been supposed to have a favourable action in Graves's disease; the enlargement of the gland in this condition, and the rarity of the disease in childhood, while the thymus persists, being cited as a rational basis for the treatment. Administration of thymus has also been recommended in rheumatoid arthritis.

Other organs and mixed extracts.—

The pancreas is an instance of an internally secreting gland the administration of which seems to have no influence on the pathological defect seen in diabetes. The attempt to stimulate the atrophic organ by giving an acid extract of duodenal mucous membrane, containing the specific pancreatic stimulant "secretin," has met with no better success.

Various other organs, not known to produce any hormone, such as the spleen and lymphatic glands, have been, and probably will be for some time, administered for a variety of conditions. With these may be classed red bone-marrow, which has been used in the treatment of anæmia, apparently on account of its hæmopoietic function in life. Apart from the fact that there is usually no evidence of defect of this function in the conditions to which the treatment has been applied, it is difficult to imagine that the ingestion of marrow from an ox or sheep could affect the production of red corpuscles in the marrow of the patient.

A good deal has been heard during recent years of the so-called "pluriglandular" therapy, which is based on the obvious though imperfectly defined functional connexion between such organs as the thyroid, suprarenal and pituitary glands on the one hand, and the sexual glands on the other. While it would seem logical, and has in practice proved efficacious, to treat a retarded sexual development, due to thyroid deficiency, by administering thyroid-gland substance, there is not much to be urged against the combination of thyroid with the appropriate gonad substance, which some would advocate in such cases. It can

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only be suggested that there is little evidence that the addition of the gonad improves the effect of the thyroid substance, and that, when once the safe ground of experimentally-proved efficacy is abandoned, free play is given to the superficial reasoning by facile analogy which has played all too large a part in organotherapy. Along this road we arrive very easily at the treatment of various vague neuroses with the mixed extracts of different ductless glands, no one of which can be clearly shown to have any relation to the condition. Such treatment has no claim to be regarded as scientific, however great the satisfaction its apparent results sometimes give to patient and practitioner alike.

H. H. DALE.

ORIENTAL SORE (*syn.* Bouton d'Orient, Aleppo Sore, Delhi Boil, Scinde Sore, Frontier Sore, Uchut, Yalek, etc., in the Old World; Espundia, Uta, Buba, etc., in South America).—A skin disease caused by invasion of the superficial layers of the skin by a flagellate, *Leishmania tropica*, which bears a very close resemblance to the parasite producing the systemic disease kala-azar.

Etiology. The organism is an egg-shaped structure measuring about 3 by 2 microns. It contains two nuclei—a large trophonucleus and a smaller and more deeply-staining rod, the kinetonucleus; longer cigar-shaped or rounded forms are sometimes seen. Cultivated in blood-agar (N.N.N. medium), there develops an elongated organism with a single long anterior flagellum.

The disease is transmitted most probably by some biting fly, and recent work points to the sand-fly (*Phlebotomus*), the well-known vector of sand-fly fever. (Fig. 79, p. 539.)

Distribution. In the Eastern hemisphere the disease occurs in Egypt, Sudan, Mediterranean littoral, North Africa, North-West India, Persia, Caucasus, Turkey, Mesopotamia, and Arabia; and in South America, in Mexico and the northern half of the Southern continent.

Clinical course.—The incubation period varies from ten days to six months or longer. The first noticeable lesion is a small papule, generally on an exposed surface, as the face, hands, or feet. It is usually regarded as an insect bite, but instead of disappearing it increases slowly but steadily in size, becomes more raised, is of a dull-red colour, and covered by thin scaly epidermis. It reaches a diameter of half to one inch and then gradually shrinks

and dries till merely a scab remains. If this is removed before healing is complete, a shallow ulcer showing pale bleeding granulations is produced. If left undisturbed, the scab eventually falls off, leaving a thin superficial scar, which eventually becomes white. The cicatricial tissue has little tendency to produce deformities by contraction. The scars are very common on the faces of those who live



Fig. 67.—Oriental sore. (After Wenyon. From photo by R. McKay.)

The sore over the nose is of the granulomatous, and that near the outer canthus of the eye is of the punched-out type.

in endemic areas, such as Bagdad and Aleppo, where they receive local names, e.g. "date mark" in Mesopotamia. The type of sore just described, sometimes called male variety by the natives, is the non-ulcerating form. More frequently the papule commences to ulcerate through the removal of the superficial epithelium (female variety). Secondary bacterial infection takes place, and there results an ulcer with raised red skin around it. (Fig. 67.) It tends to form a scab which encloses a purulent exudate. The ulcerating varieties are more

extensive and produce more scarring than the non-ulcerating forms. They do not become adherent to deeper structures, and are generally painless, unless in a region such as the elbow, where skin is stretched over bone. Itching is a common feature, while the exudate may be very offensive, probably as a result of the secondary bacterial infection. There are practically no constitutional symptoms. Left to run a natural course, the disease lasts 6-18 months. The sore is frequently single, but very commonly there are two or three. A larger number is occasionally seen. There may be as many as a couple of dozen on the face alone, or they may be distributed about the body.

In South America (and very rarely in the Old World) the benign skin lesions just described may be followed by invasion of the mucous lining of the mouth and nose. Extensive erosions and overgrowths of the soft parts occur, and this condition, lasting in some cases for many years, reduces the subject to a condition of profound cachexia.

Diagnosis.—In making a diagnosis the long history of the case and the locality are of importance. It is established by discovery of the organism, and this is done by examination of smears made from scrapings of the sore, or better still, in order to avoid the secondary bacteria, from material obtained by deep puncture, with a fine glass pipette, of the raised red skin around the ulcer. The films are stained by Leishman's stain or some other modification of the Romanowsky method. Apart from the finding of the parasite, diagnosis is difficult, as the condition may be confused with syphilis, rodent ulcer, lupus, furunculosis, yeldt sore, Nile boil, and other chronic ulcerations. Sometimes the organism has been recovered by culture when direct observation of smears has failed. Glandular enlargement in associated gland areas may occur, and the parasite has been found in such glands.

Treatment.—The older method of excision is now rarely practised, for unless a wide margin of healthy skin is removed parasites are left behind and relapse will certainly occur. Excision furthermore increases the scarring. The latter statement is also true of treatment by escharotics, such as permanganate of potash applied in powder form, nitric acid, carbolic acid, and similar agents. Carbon-dioxide snow in pencil form has been employed. Ointments of tartar emetic (2 per cent.) or methylene-blue (equal parts of methylene-blue, lanolin, and

vaselin) are less irritating, though healing does not commence till after about a fortnight's daily application of the ointment is stopped.

The best treatment is undoubtedly that by intravenous injections of tartar emetic (potassium antimony tartrate) or of the corresponding sodium salt. A 1-per-cent. solution is used and a dose of 10 c.c. is injected once, twice, or even thrice weekly. Two or three weeks' treatment is usually sufficient. The 10 c.c. may be further diluted to 50 or 100 c.c. before injection.

Good results have also been reported from ionization. Ten thicknesses of lint soaked in 1-in-1,000 to 1-in-500 perchloride of mercury solution are placed over the sore. The positive pole of a 10-150 millimetres ampere current is employed. Two exposures of fifteen minutes each are required. Chlorine ionization has also been successfully employed.

Left untreated, and protected from irritation and injury, the sore will heal naturally in about a year, leaving behind an immunity from further infection.

C. M. WENYON.

OSTEITIS (see EPIPHYSITIS; OSTEO-MYELITIS; PERIOSTITIS).

OSTEITIS DEFORMANS (*syn.* Paget's Disease of Bones).—A condition in which certain bones become enlarged and altered in shape and structure.

Etiology.—The cause is unknown, though many cases give a positive Wassermann reaction. The disease begins in middle age, is occasionally hereditary, and is seen in men much more frequently than in women.

Pathology.—The bones affected are the long bones—especially the femur, tibia, clavicle, fibula, and ribs—the spine, and the skull bones. The cancellous bone becomes rarefied and even cystic, whilst under the periosteum new bone is deposited, so that the affected bone becomes enlarged in length and breadth. The long bones become exceedingly curved, those of the lower limb being bowed forwards and outwards, with associated coxa vara. The external aspect of the skull is thickened, and the cervical and dorsal spine develops marked kyphosis.

Symptomatology.—The disease begins in one bone, particularly the femur, clavicle, or tibia; there is chronic aching pain, sometimes giving a sensation of tension, increased by fatigue; involvement of the skull causes

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severe headache. Deformities develop, and the attitude becomes simian; the upper limbs may be lengthened, the lower limbs are shortened, and the gait becomes waddling, with legs apart; there is a sense of weakness and lack of balance, necessitating the support of a walking-stick. In the back a marked cervico-dorsal kyphosis develops, and the normal lumbar curve disappears. Loss of stature results, and the patient has to take a larger size in hats. Spontaneous fracture may be the first thing to call attention to the disease; not uncommonly a fracture is due to the supervention of endosteal sarcoma.

Diagnosis.—When only one bone is affected it is difficult to diagnose osteitis deformans from *syphilis*; the X-ray appearances (see X-RAYS, DIAGNOSTIC USES OF) and the progression of the osteitis in spite of treatment, will settle the question. *Acromegaly* is distinguished by its symmetry and the marked changes in the face, hands, and feet. *Osteomalacia* affects especially the lumbar spine and pelvis; its association with pregnancy and the absence of a waddling gait are distinctive. *Osteitis fibrosa*, usually occurring first in the tibia in young adults, closely resembles osteitis deformans pathologically.

Prognosis.—There is no known means of arresting the slow progress of the disease, which lasts about twenty years, and is terminated by the development of sarcoma, or by a complication of a fracture, such as hypostatic pneumonia.

Treatment.—Antisymphilitic treatment may be applied if the Wassermann reaction is positive, but it does not produce striking results; otherwise treatment is directed to relief of pain by avoidance of fatigue, the application of radiant heat, and the use of aspirin. Risk of slight traumata must be avoided.

C. W. GORDON BRYAN.

OSTEO-ARTHRITIS.—A chronic joint-disease, usually affecting elderly subjects, and characterized anatomically by marginal osteophytic outgrowths from, and central eburnation and erosion of, the articular surfaces. The condition is monarticular or oligarticular rather than polyarticular. Large joints are affected rather than small, and if more than one joint is involved the distribution is not strikingly symmetrical.

Etiology.—(1) There is probably a large class of *purely degenerative cases*. Just as a man's arteries sclerose with advancing years,

so also his articular cartilages become brittle and more susceptible to the effects of wear and tear, resulting in the eburnation and erosion characteristic of osteo-arthritis.

(2) Another group, sometimes called the static group, is *secondary to deformities* such as pes planus or genu valgum, whereby the body-weight is transmitted to the ground along abnormal lines of force, and consequently the feet, knees, or hips are subject to abnormal stresses.

(3) Some cases are undoubtedly due to *trauma*, and most of the rare instances of this condition in young subjects are so caused. A comparatively trivial injury to an exposed joint such as the metacarpo-phalangeal joint of the thumb is sufficient to set up a chronic osteo-arthritis of this joint. Possibly the osteo-arthritis often seen in gouty subjects is really traumatic, the repeated deposition of uric acid in the articular cartilage acting as an injury. Exposure to damp and cold is certainly a predisposing factor.

(4) *Sepsis*.—It is possible that some cases may be caused by absorption of organisms or their toxins from some chronic septic focus, e.g. the gums or colon. Experimentally, the characteristic morbid appearances of the disease have been produced by the injection, intra-articular or intravenous, of streptococci or other organisms.

Age.—Osteo-arthritis is a disease of the elderly, being most frequent in patients over the age of 40, but is occasionally seen in young subjects.

Sex.—Men are more often attacked than women, possibly owing to the greater exposure of the former to cold, injury, and wear and tear. Women are particularly prone to develop osteo-arthritis of the knees during the climacteric, but their hips and shoulders are not nearly so liable as those of men.

Pathology.—As a rule only one, or at most three or four, joints are affected, the large joints, especially the knees, hips, and shoulders, being more often involved than the small. The smaller joints, however, may be the seat of the disease, which is by no means uncommon in the metacarpo-phalangeal joint of the thumb, and in the metatarso-phalangeal joint of the great toe, where it is usually associated with hallux valgus. Polyarticular osteo-arthritis may undoubtedly occur, but its clinical differentiation from the end-results of chronic septic arthritis is exceedingly difficult. Osteo-arthritis is a disease of the articular

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ends of the bones rather than of the synovial membrane. The cartilage loses its pearly lustre, becomes less elastic and fibrillated, and is eventually eroded where the pressure is greatest; the exposed bone being converted into a hard, ivory-like substance ("eburnation"). At the margins of the articular surface the cartilage proliferates, either more or less evenly along its periphery ("lippling"), or locally into knob- or spike-like excrescences, which on ossification provide the osteophytes. The joint-movements may be limited, or even entirely abolished, by the interlocking of these osteophytes, but no true ankylosis occurs. Small fragments of cartilage or of synovial fringes may become detached and form free bodies in the joint-cavity. There may be an excess of synovial fluid in the joint-cavity, especially after a slight trauma or when a synovial fringe or a free body has been nipped between the articular surfaces. This fluid is usually quite clear and sterile. As a rule the changes found in the synovial membrane are scanty, as contrasted with those of the articular surfaces, but in some cases a remarkable proliferation of the membrane produces a cauliflower-like appearance of this structure. This condition, known as the "villous joint," is usually secondary to some static defect, such as flat-foot, and is most common in the knees, which are often symmetrically affected.

Collections of encysted fluid that are known as *Morant Baker cysts* are sometimes found in the tissues adjacent to the joints, especially the knee and hip. They may be so large as to impede the joint-movements and to require excision on this account.

Symptomatology.—Pain in most cases first brings the patient to the doctor, but the amount of pain felt is extraordinarily variable. It is by no means uncommon to discover more or less accidentally a very gross osteo-arthritis creaking of the knees which has been unassociated with any symptoms whatever. On the other hand, there are few more painful conditions than severe osteo-arthritis of the hip. The pain is frequently referable to changes in the weather, and, oddly enough, very hot dry weather is often almost as distasteful to these patients as is cold and damp. The affected joints are very susceptible to slight wrenches or strains, which may easily bring about an exacerbation of pain, associated perhaps with a slight and transient effusion into the joint. The pain may be referred to an area away from the joint. Thus, hip-joint

pain may be referred to the knee or along the course of the sciatic nerve, so that osteo-arthritis of the hip is not infrequently incorrectly diagnosed as sciatica.

Limitation of joint-movements is common, and may be due to muscular spasm consequent on the pain induced by attempted movement, or to interlocking of osteophytes. As has been said above, true ankylosis never occurs. The joint is always most "stiff" at the beginning of the day, and the range and freedom of movement improve after the first attempts at movement. The onset is commonly very insidious and the course of the disease very slowly progressive, with perhaps the temporary exacerbations of symptoms and signs resulting from trivial injuries or strains.

The **diagnosis** depends partly on the symptoms and partly on the physical signs; the most important of the latter is the characteristic crepitus produced on joint-movement. In the early stages the crepitus may be very fine, and obtainable only by careful palpation. When there is gross destruction of the articular cartilage, a coarse grating or creaking may be produced, which is audible as well as palpable. In the case of superficial joints, e.g. the metacarpo-phalangeal joint of the thumb, the marginal osteophytic thickening may be both palpable and visible. As a rule there is little, if any, synovial thickening, and an obvious excess of synovial fluid is uncommon except during exacerbations produced by strains or by gross over-use of the diseased joint. As an exception to this rule must be mentioned the "villous joint" previously referred to, in which there may be great synovial thickening, and possibly also excess of fluid.

There is but little pain on palpation of the joint, though there may be a little tenderness along the "joint-line." The joint can often be moved passively through a considerably wider range than it can be actively, unless movement is limited by interlocking osteophytes, which interlocking can be plainly felt.

Wasting of the muscles supplying the joint is an early phenomenon, and is often very pronounced, but is rarely so extreme as in rheumatoid arthritis, nor are contractures so common as in the latter condition.

The general health is not impaired, unless pain is so severe as to interfere with sleep and to cause mental depression. There is neither fever nor sweating, and no enlargement of the spleen or of lymphatic glands; trophic changes of the skin or nails are not observed.

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The X-ray findings in osteo-arthritis are characteristic. Though the central areas of the cartilage are eroded, elsewhere the cartilage is at least of normal thickness, and consequently, in the radiograms, the "joint-slits" are well preserved. Marginal lipping and possibly osteophytes are commonly present; there is no great rarefaction of bone such as is constant in rheumatoid arthritis. (Plate 43, Fig. 3, Vol. III, facing p. 550.)

Osteo-arthritis of the hip (*malum coxae senilis*) is often diagnosed as *sciatica*, the ultimate diagnosis sometimes depending on the X-ray findings. In the joint-disease, pain may be referred down the sciatic nerve, but not below the knee, muscular wasting is more pronounced, but confined to the buttock and thigh, thickening of the joint and crepitus may be detected on palpation, and the patient is often unable to sit with legs crossed.

Heberden's nodes are little round knobs due to small osteophytic outgrowths on each side of the bases of the terminal phalanges. Sooner or later after their appearance follows a more or less pronounced lateral deflection of the terminal phalanx, and not infrequently slight osteo-arthritic symptoms and signs are observed in other joints. These nodes often appear in women after the climacteric, but are by no means uncommon in men. The associated osteo-arthritis is usually of a comparatively benign type.

Charcot's joints differ from osteo-arthritis in being comparatively painless. There is much more destruction of the articular surfaces, and at the same time there may be considerable osteophytic outgrowths; large effusions of fluid, and dislocations or other gross deformities, are the rule. The diagnosis will be clinched by the finding of evidence of tabes or of syringomyelia.

Prognosis.—Though individual joints may in time become seriously disorganized, the patients can always be assured that at most the course of the disease is likely to be exceedingly slow, and that it will not end in widespread deformities and crippling. Many cases of well-marked osteo-arthritis of the knee or shoulder never cause more trouble than occasional bouts of pain or swelling of the joints, and a certain degree of limitation of movement. Osteo-arthritis of the hip, on the other hand, may be an exceedingly painful condition, and may give rise to very grave disability.

Treatment. General.—When the knees or hips are affected, deformities such as pes

planus should be looked for and, when present, corrected if possible. A search must be made for chronic septic foci, particular attention being paid to the nose and its sinuses, the mouth, the ears, the alimentary canal, the respiratory tract, the vagina, the bladder, and the urethra; if any pathological state is found it should be dealt with appropriately.

The general health should be maintained at its optimum by means of a generous diet; there is no need for dietetic restrictions unless there are obvious indications of a gouty element. The weight of obese patients should be reduced by suitable measures, especially if the joints of the legs are affected.

Residence in a dry and equable climate is desirable, and it is very important that the affected joints, even when the disease is quiescent and giving rise to no symptoms, should be warmly wrapped up and protected from cold.

Drugs are not of much avail, though a simple tonic mixture is often helpful. Pain may best be relieved by aspirin; it is important that this drug, or even morphia, should be administered at night if pain is interfering with sleep. The combination of salicin with potassium iodide is often useful, as in the following formula:—

17 Salicini gr. x.
Pot. iod. gr. v.
Syr. glycerophos. co. ʒi.
Aq. ad ʒi.
Ft. mist. 1 oz. t.d.s., p.c.

Local treatment.—Hot-air baths, or radiant heat, followed by massage and cautious passive movements, are very successful in this condition, the relief of pain and increased freedom of movement after their use being quite remarkable. Ionization is also successful sometimes, though it is probable that the hyperæmia produced by the passage of a sufficiently heavy current is a factor more important than the introduction of any particular ions. Scott's dressing is a useful counter-irritant.

So long as there is any considerable pain in the joint, and especially if there is any swelling or heat, it should be kept at rest, if necessary by a splint. It is a great mistake to attempt to keep the movements of the joint free by excessive use. In the case of the knees and hips, brilliant results have sometimes been obtained in carefully selected cases by the use of a Thomas calliper splint, or of Hoeffcke's splint.

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Finally, in certain cases, surgical measures are called for. Interlocking osteophytes may be chiselled away, or more or less extensive resections of the joint structures may be undertaken, sometimes with very gratifying results.

MAURICE CASSIDY.

OSTEO-ARTHROPATHY, PULMONARY (*syn.* Hypertrophic Osteo-arthritis).—Symmetrical enlargement of the hands and feet, with clubbing of the fingers and toes, periostitic thickening of the long bones, especially in the neighbourhood of the joints, and articular inflammation (H. D. Rolleston).

Etiology.—The disease occurs more often in males than in females. It is almost always secondary to pulmonary disease, and has most often been found associated with empyema, bronchiectasis, chronic pneumonia, phthisis, or malignant disease of the mediastinum, pleura, or lung. Rarely, the initial disorder has been Hanot's cirrhosis of the liver, spinal caries, or intrathoracic lymphadenoma; occasionally no primary lesion is discoverable. The condition is probably produced by toxins absorbed from the antecedent disease, and therefore usually from the lungs.

Pathology.—The essential lesion is a new formation of periosteal bone on the shafts of the long bones, especially in the neighbourhood of joints. The lower ends of the radius and ulna, the metacarpal bones and the first and second rows of phalanges are most commonly affected, but the bones in the neighbourhood of the elbow- and ankle-joint sometimes show the same changes. The new bone may extend beneath the periosteum along the shafts of the long bones, but is less and less thick the farther it is removed from the joint. The joints themselves may contain fluid, and their synovial membranes may be thickened.

Symptomatology.—To enlargement of the ends of the bones and swollen joints is added clubbing of the fingers, which is constant and often extreme. Sometimes the joints become inflamed, tender, painful and restricted in movement, with redness of the overlying skin. The exacerbations are accompanied by fever. The enlargement near the joints is not due exclusively to the new bone-formation, for the adjacent subcutaneous tissue is swollen in the same manner as the finger-tips. X-rays assist the diagnosis by demonstrating the new periosteal bone, under which the shaft is rarefied. Somewhat similar changes are seen in a radiogram of osteo-periostitis, but the

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subadjacent rarefaction is distinctive. The clubbing of the fingers leaves little doubt as to the true diagnosis.

Treatment.—The treatment should be directed to the primary disease: when this can be eliminated, as in cases of empyema, the osteo-arthritis gradually disappears. The malady seldom needs individual treatment. During an exacerbation, resting the affected joints, with the application of heat and administration of opium, affords relief.

FREDERICK LANGMEAD.

OSTEO - CHONDritis (*see* PSEUDO-COXALGIA).

OSTEO - CHONDritis DEFORMANS JUVENILIS (*see* PSEUDO-COXALGIA).

OSTEOGENESIS IMPERFECTA.—A rare condition in which multiple fractures of bones occur in intra-uterine or infantile life.

It is convenient to regard *fragilitas ossium* as including (1) *osteogenesis imperfecta* and (2) *mollities ossium* or *osteomalacia* (q.v.). These are two separate diseases. In *osteogenesis imperfecta*—for which condition the terms *annular rickets* and *periosteal aplasia* have also been suggested—the fractures are primary and the deformities and bending secondary. In *osteomalacia* the softening is primary and the fractures are secondary features (H. D. Rolleston).

Etiology.—In about a quarter of the cases the disease is hereditary. The ossification from periosteum is defective.

Symptomatology.—The tendency to fractures may arise in utero, or may start in the first few months after birth. Occasionally the onset of the disease is delayed until late childhood. The ribs and the long bones of the extremities suffer most, but the fractures are not confined to them. The fractures occur from very slight injury or even apparently spontaneously, being possibly due to muscular effort. They are attended with little pain, and are often very numerous. Blueness of the sclerotics has been noted in some cases.

Diagnosis.—As a rule the disease is easily distinguished from rickets or osteomalacia.

Prognosis.—Starting in intra-uterine life, the fractures usually cause the infant to be delivered dead. Arising soon after birth, the disease is often fatal, but in later life has little tendency to kill. After a few years the tendency to fracture diminishes or disappears. Osteomalacia may supervene. The fractures rarely fail to unite, but the union is of calcified

fibrous tissue and not osseous. Much callus-formation is common, and deformity is very pronounced.

Treatment.—In severe cases fractures are practically unavoidable, nor can the usual methods of treating them be adopted. The limb should be wrapped in wool with a soft bandage and left. As the disease passes off, operations may be undertaken to correct the extensive deformities.

REGINALD MILLER.

OSTEOMALACIA.—Osteomalacia, also called mollities ossium, is a form of fragilitas ossium (see OSTEOGENESIS IMPERFECTA) in which certain bones bend and are subject to spontaneous fracture as a result of the absorption of their mineral salt elements.

Etiology.—The disease is very much more common in women than in men, and usually begins during pregnancy; it attacks persons whose diet is deficient.

Pathology.—The bones of the lumbar spine and pelvis are most affected; decalcification leads to frailness and a tendency to flexibility, spontaneous fracture, and deformity. The marrow and cancellous bone are soft and vascular, and the periosteum is thickened. Calcium salts are excreted from the system in excess. The pelvis becomes narrowed and triradiate from the pressing of the acetabula inwards and of the pubis forwards, while the sacrum bends forwards.

Symptomatology.—The disease usually starting during pregnancy, its most important result is difficulty in parturition. There is indefinite pain in the affected regions, with general wasting and weakness; in non-pregnant women the symptoms are exaggerated during menstruation. Deformity develops; the sternum and symphysis pubis are prominent, the chest is flattened from side to side, with displacement of the arms upwards and backwards, and the distance between the great trochanters is decreased; there is lumbar lordosis, and genu valgum may occur; when erect the patient leans forward and supports herself on her hands. Spontaneous fractures may occur, and are followed by weakness or absence of union.

Prognosis.—The disease usually progresses, with intermissions, until the patient becomes bedridden, and eventually dies from wasting and exhaustion; but it is sometimes arrested at the menopause, natural or artificial. There is grave danger of death during childbirth.

Treatment is directed to the relief of pain

by aspirin, salicylates, and the application of radiant heat, and calcium salts are administered. In pregnant women the danger of full-time natural delivery must be prevented by the induction of premature labour, or by Cæsarian section combined with removal of the ovaries. If the disease occurs in a non-pregnant woman, oöphorectomy should be performed, unless the natural menopause is imminent.

C. W. GORDON BRYAN.

OSTEOMATA (see EXOSTOSES).

OSTEO-MYELITIS.—Inflammation, either acute or chronic, affecting all the component parts of a bone. In this place osteo-mylitis due to pyogenic bacteria is described; the forms due to syphilis and tuberculosis are dealt with in the articles on BONE, SYPHILIS OF, and BONE, TUBERCULOSIS OF.

Etiology.—The causative bacteria are, in order of frequency, *Staphylococcus pyogenes aureus* and *albus*, *Streptococcus pyogenes*, the pneumococcus, and *Bacillus typhosus*. General debility, especially if due to loss of blood, is a predisposing cause. The infection generally reaches the bone by means of the blood-stream, and there is often a history of injury to the bone; this variety usually occurs in children, and is more common in boys than in girls, because the former are more liable to injuries. In other cases the disease is the result of septic infection of a compound fracture, the bacteria gaining access through the wound. Lastly, the bone may be infected by direct spread from an adjacent focus in a joint or in the soft parts.

Pathology.—In blood-stream infection the primary focus is usually in the skin, a furuncle, carbuncle, or septic abrasion, or it may be in the tonsil or other area covered by mucous membrane.

The bone-infection most often starts in the centre of the cancellous tissue at the end of the diaphysis, but may begin in the epiphysis, in the deep layers of the periosteum, or occasionally, if due to streptococci, in the medullary cavity; in any case, a preceding slight injury may have produced a small hæmatoma in the bone, and in this the organisms are deposited. The bones most often infected are the tibia, the humerus, and the femur; in the femur it is common for the disease to begin as a periosteal infection of the lower part of its posterior surface.

The inflammation rapidly spreads through the cancellous tissue to the medulla; the in-

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creased tension leads to thrombosis of the vessels of the bone, and necrosis of a large part of it follows; the periosteum is detached, and an acute abscess forms under it and rapidly tracks along the bone.

It is common for the inflammation to spread to the neighbouring joint, which becomes distended at first with clear fluid, later with pus, all the changes of suppurative arthritis supervening; in young children, especially, the joint is affected early.

The dead bone forms a large sequestrum, and becomes enclosed in an involucrum of new bone, regenerated from the inner surface of the inflamed periosteum; the involucrum is lined by granulations that discharge a large amount of pus, which escapes through holes, "cloacæ," in the involucrum. In some very acute cases no involucrum is formed. Either as a result of natural processes or of operation, sinuses form in the skin and lead down through the cloacæ to the sequestrum; they become lined with prominent granulations and there is profuse purulent discharge. If the sequestrum is removed early, the cavity in the involucrum becomes obliterated by bone-regeneration, but if the sequestrum is allowed to remain until the involucrum has become sclerosed, the power of osteogenesis is lost and the cavity persists permanently, the sinuses continuing to discharge for an indefinite length of time. Until it is firmly ossified the involucrum is liable to fracture or bending.

In some cases the disease runs a less acute course and a chronic abscess forms in the interior of the bone, surrounded by a zone of sclerosis and usually containing a small sequestrum; the name *Brodie's abscess* is given to this condition, which may persist for years without severe symptoms; such abscesses are sometimes multiple.

As a result of detachment of the septic thrombi in acute osteo-myelitis, pyæmia may occur and give rise to foci of suppuration in other parts of the body, especially joints, tendon-sheaths, and bursæ. As a late result of chronic suppuration, lardaceous disease occasionally supervenes.

When osteo-myelitis is the result of direct infection of a compound fracture its course is usually less acute, as the fractured surface allows drainage of the bone; tension thrombosis is therefore not a feature, and extensive necrosis does not occur. Sequestra may result from comminuted fragments which have had their periosteal attachments severed by the

original injury or by operative procedures. In infected open amputations from which the sawn end of the bone protrudes, osteo-myelitis leads to a conical sequestrum which takes a very long time to separate.

Symptomatology.—Several clinical forms occur, according to the acuteness of the disease. In the most virulent the general symptoms are so severe that they may mask the local ones; the temperature is high, the pulse rapid, the patient is delirious or comatose, and early death results from the intense toxæmia.

In another form the symptoms are those of pyæmia, with local signs related to the diseased bone; rigors and the development of abscesses in other parts of the body, especially in the hip- and shoulder-joint and under the deep fascia of the back, are the most prominent features, and death from toxæmia or the involvement of an important viscus is very liable to occur.

In the form which is fortunately the most common, the main symptoms are those of the local disease combined with fever. The onset is acute, and severe aching pain in the affected limb is complained of, sometimes actually localized in the diseased end of the bone; there is pain on touching or moving the limb, elicited in the earliest stages by pressure or percussion of the bone at a distance from the site of the inflammation. The neighbouring joint becomes tender and swollen, with limitation of movements; at first the effusion is aseptic, but later it becomes infected and suppurative arthritis ensues. The skin over the affected bone is red, hot, and ardematous, especially if the bone has a subcutaneous surface; with a deeply placed bone there may be cedema of the limb some distance away. The temperature is high and the pulse rapid, the face is flushed and the tongue furred. Delirium is not uncommon, and death may occur from toxæmia and exhaustion. In the less virulent cases a subperiosteal abscess forms, over which the skin becomes red and fluctuant; the abscess may even burst through the skin with some relief of the symptoms. In such cases the disease then settles down to a chronic course, but this sequence of events is unusual, and in most instances only prompt surgical intervention will save the patient's life. A high leucocytosis is present while there is pus under tension.

In the *chronic stage* which follows surgical or spontaneous evacuation of the pus the limb

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becomes thickened, partly from brawny oedema of the soft parts, partly from the development of the involucrum; usually several sinuses are present, lined by prominent granulations and leading through the cloacæ to necrosed bone, with a copious discharge of thick pus. After months or years, diarrhoea, wasting and albuminuria may indicate the onset of lardaceous disease.

In some cases of mild infection, after an acute onset the inflammation settles down to a chronic *abscess*, which may persist for years and give little sign of its presence; as a rule there is aching pain, especially at night or after exercise, with tenderness on deep pressure, and slightly increased heat, and oedema of the overlying skin. The patient is subject to slight fever at night and feels "shivery" at times, and exacerbations of acute pain and fever may occur. X-rays show a local area of rarefaction surrounded by sclerosis, and a small sequestrum is often present.

In enteric fever, subacute osteo-myelitis of a rib sometimes develops in the later weeks of the illness, or even a long time after recovery from the acute disease; the affected bone becomes painful and thickened, and a subperiosteal abscess commonly results.

Osteo-myelitis of a compound fracture is characterized by an intermittent temperature, profuse discharge from the wound, and, by X-ray examination, an appearance of rarefaction of the central part of the bone, extending from the fracture; there is no surrounding sclerosis in the earlier stages, and necrosis does not usually occur if the wound is open. If, however, the wound has been closed without drainage, or if the bone has been wired or plated, the pus tracks and the periosteum is stripped up, with resultant necrosis of bone.

Diagnosis.—The most important conditions for which acute osteo-myelitis might be mistaken are other forms of septiciæmia, rheumatic fever, enteric, or other acute specific infection; injury to bone, such as incomplete fracture or traumatic periostitis; and gonococcal or other form of acute arthritis.

The cases of osteo-myelitis which simulate an *acute specific fever*, especially enteric, are those in which the general symptoms, particularly delirium and coma, are so intense that the local manifestations are masked; in all such cases a systematic examination of all the bones must be made for tenderness or other sign of acute inflammation; the prognosis in these cases is extremely bad, and even early

amputation rarely prevents rapid death. In *injuries* there may be some bruising, the tenderness is quite localized, there is little or no fever, and there is no leucocytosis. *Acute rheumatism* of one joint only is unusual in young people, whereas acute osteo-myelitis is a common disease; in arthritis pain only occurs when the joint is moved, and the general symptoms of toxæmia are less severe in the early stages, nor is there such a high leucocytosis. When in osteo-myelitis the symptoms and signs are chiefly due to an accompanying arthritis of the neighbouring joint, it would be a serious matter to diagnose acute rheumatism and to postpone surgical intervention while treatment by salicylates is carried out. In view of the rarity of acute monarticular rheumatism in the young, the rule must be to treat all cases of acute arthritis of one joint in children, if accompanied by high fever and toxæmia, as osteo-myelitis of the end of the neighbouring bone, especially if it is the shoulder, the hip, or the knee that is affected, unless, of course, another obvious cause for the arthritis is present. Similarly, the symptoms of acute osteitis of the astragalus will be those of *acute arthritis of the ankle*.

Gonorrhœal arthritis usually affects adults, and the discovery of the primary focus, the localization of the inflammation to the joint and the periarticular soft parts, and the absence of severe general symptoms are distinctive.

In distinguishing typhoid osteitis of a rib from *tuberculosis*, the history of the preceding acute illness and the Widal reaction are the important features, or the pus may be aspirated and examined bacteriologically.

Brodie's abscess has to be diagnosed from *gumma*, from *localized periostitis*, and from an *endosteal growth*. The question will usually be settled by an X-ray examination showing the rarefied area surrounded by sclerosis, and often containing a small sequestrum; in doubtful cases an exploratory operation must be undertaken. (Plate 42, Figs. 1, 2, Vol. III, facing p. 547.)

Prognosis.—In the most acute cases, giving rise to delirium and coma, recovery is rare; in all cases there is grave risk to life from pyæmia. If recovery ensues, the patient's relatives must be warned that months or years of treatment may be necessary, with more than one operation, and that deformity from defective growth of the bone is liable to follow, especially if the disease is in the neighbourhood of the knee, shoulder, or wrist; very occasionally overgrowth of the bone is a result.

Treatment.—The *immediate* treatment must be operative; an incision through the soft parts and the periosteum is made and the compact tissue is removed so as freely to open up the medulla and particularly the cancellous tissue at the affected end of the diaphysis. At the end of the operation the wound is lightly packed with gauze soaked in liquid paraffin containing 1 per cent. iodoform.

In the *after-treatment*, careful splinting is very important, to prevent pain, auto-inoculation, suppurative arthritis, and the risk of fracture or bending of the involucrum; some form of splint, such as the Thomas knee-splint, which allows access for dressing without disturbance, is the best.

After the first two days the best means of applying an antiseptic is *Carrel's method* of intermittent moistening with eusol or Dakin's solution, introduced through multiple rubber tubes fixed in the wound, which is lightly filled with gauze renewed daily. The pain of dressing may be minimized by covering the raw surface with a sheet of thin perforated rubber tissue placed between tubes and gauze; in toxæmic cases frequent anæsthetics for dressing should be avoided. The Carrel treatment should be continued for about four days; when the surfaces begin to granulate, the iodoform-paraffin dressing is reverted to. The early use of vaccines has given good results in some cases.

As *general treatment*, plenty of fluids are given by the mouth, and in the early stages by the rectum in the form of saline or water; subcutaneous saline should, if possible, be avoided, because of the risk of infection being conveyed by the blood-stream to the site of the injection. Tonics, particularly iron, are valuable in the later stages, and the patient should be moved into the open air as much as possible, especially if sunlight is available.

If necrosis of the shaft of the bone occurs, as it does in most cases, the sequestrum must be removed. Operation should be performed for this purpose as soon as the limits of necrosis are defined, usually about the sixth week; if this is postponed, the involucrum becomes sclerosed and loses its powers of osteogenesis, so that the granulation-lined cavity persists, and sinuses remain unhealed for an indefinite period. X-rays will help in deciding the outline of the sequestrum.

In cases in which the disease is so acute and extensive that an involucrum is not formed, deformity may be considerable, especially in regions where there is no parallel bone

to preserve the length of the limb; such a consequence can be avoided by careful extension-splinting. The Thomas knee-splint, with fixed extension applied to a transfixion pin placed above the os calcis, is the most convenient method in osteo-mylitis of the femur or the tibia. Splinting must be continued till the newly regenerated bone is strong; in later stages plaster-of-paris may be employed. Little weight must be transmitted through the bone till X-rays show it to be of normal density, but the patient may be allowed to walk on a calliper splint which transmits his weight from the tuber ischii to the heel of his boot.

Various operative measures are adopted to promote healing; when sinuses persist from the existence of a permanent cavity in the new bone, the removal of two-thirds of its wall, inversion of skin flaps, and the use of fat, muscle, or bone-grafts may be necessary.

An accompanying arthritis must be treated on the lines indicated in ARTHRITIS, ACUTE INFECTIVE.

C. W. GORDON BRYAN.

OTIC NEURALGIA (see NEURALGIA).

OTITIS EXTERNA.—Under this designation are included diffuse external otitis and circumscribed external otitis, or furunculosis of the external auditory meatus.

1. ECZEMA OR DERMATITIS OF THE AURICLE AND EXTERNAL MEATUS.—The most frequent cause is purulent otorrhœa, but it may arise independently of any local cause.

The **symptoms** are those of eczema elsewhere. When chronic, the infiltration may be so considerable that it may lead to serious narrowing of the meatus, and to partial retention of the discharge, a state of things which can only be remedied by a mastoid operation.

The **treatment** of eczematous dermatitis due to discharge consists first of all in the removal of the cause, but the skin disease itself should also be treated. If it be acute, the application to the auricle of lint soaked in dilute lead lotion is advisable, while the following drops may be inserted in the canal:—

℞ Plumb. acetat. gr. xxiv.
Sol. alumin. acetat. ʒi.
Aq. dest. ad ʒi.

If the condition be chronic, the frequent application of the following ointment will hasten cure:—

℞ Ung. hydrarg. oxid. flav. gr. vi.
Paraff. (vaselin) ʒi.

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2. SEBORRHOIC ECZEMA. — The patient's chief complaint is of itching and irritation in the canal and auricle. Discharge is scanty or absent, and scale-formation common. The condition is always associated with seborrhoeic eczema of the scalp. The most useful application is ung. acid. salicyl. Care should be taken to treat the scalp also.

3. OTOMYCOSIS. — This form of otitis is due to a fungus, the *Aspergillus glaucus* or *A. niger* being the most common. On examination of the meatus a membranous-looking cast of the canal is usually visible. The canal is often exquisitely tender, and removal of the friable cast is consequently a task of some difficulty. Microscopical examination is necessary.

Treatment consists in the careful removal of all debris under meatal inspection, aided by the application of hydrogen peroxide. After the canal has been cleared it should be mopped out carefully with argent. nit. (12 per cent.), or with absolute alcohol. Afterwards ear-drops consisting of alcohol should be instilled once or twice daily, to prevent a regrowth of the fungus.

4. FURUNCULOSIS OF THE EXTERNAL MEATUS. — A common and painful complaint, due to a staphylococcal infection of a hair-follicle, and consequently only encountered near the orifice of the canal. Successive crops of furuncles are to be expected, and both ears may be affected either simultaneously or successively.

The chief **symptoms** are pain and tenderness. The pain, which is very severe, is increased by the movements of the jaw in speaking and in chewing. When the swelling of the furuncle blocks the meatus, there is much deafness. In from two to four days the boil breaks, the pain being thereby relieved. The discharge is purulent and profuse, and a "core" has to come away before healing takes place.

The **diagnosis** between furunculosis of the external meatus and *mastoid abscess* is often difficult, especially when the furuncle is situated upon the posterior wall of the meatus, as in that situation it causes swelling and oedema over the mastoid process. The distinction rests upon two points: (1) in furuncle there is no tenderness over the mastoid process itself, whereas in mastoid abscess the tenderness there is acute; (2) in furuncle the oedema obliterates the sulcus or groove between the auricle and the mastoid region, whilst in mastoid abscess this groove is deepened. In

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furuncle also the meatal orifice is usually closed by the swelling of its walls.

Treatment. — A free incision should be made through the swollen wall in such a position that it traverses the crater of the furuncle. The knife should be carried down to the bone, and the incision should then be well curetted with a sharp spoon. This is a painful proceeding, and requires a general anæsthetic, nitrous oxide gas being the most suitable. A drain of gauze should then be inserted into the meatus and hot fomentations applied. Recurrence may be prevented by the use of antiseptic drops (sol. hydrarg. perchlor. 1:1,000, provided there is no perforation of the membrane). Glycerin should be avoided. If, in spite of local treatment, recurrence takes place and persists, an autogenous vaccine may be tried.

DAN M'KENZIE.

OTITIS INTERNA (see LABYRINTH, AFFECTIONS OF; MENIÈRE'S DISEASE).

OTITIS MEDIA. — Under this term are included acute catarrh and suppuration, chronic suppuration with cholesteatoma, and chronic catarrh, hypertrophic and atrophic.

1. ACUTE CATARRH AND SUPPURATION OF THE MIDDLE EAR

These conditions are due to infection of the middle-ear spaces by pyogenic organisms, which generally obtain access to the middle ear by way of the Eustachian tube from the naso-pharynx. Accordingly, acute otitis media is a common sequela of "colds," influenza, scarlet fever, measles, and other diseases which cause naso-pharyngeal inflammation, and is often due to adenoids. In children, otorrhœa is almost always associated with adenoids. The commonest causative organisms are streptococci, the pneumococcus, *Micrococcus catarrhalis*, and the influenza bacillus. Ear infection is mono-microbial during the acute stage, and poly-microbial if it becomes chronic.

Pathology. — According to the relative virulence of the infection, the severity of the local lesion varies from a simple sero-mucous catarrh to widespread necrotic destruction, the latter being most usually due to scarlet fever or influenza. In the catarrhal type the reaction is mild, and the exudate gathers in the general cavity of the tympanum. In the purulent types the reaction is more violent and, as the seat of most intense inflammation is in the attic or upper part of the tympanum among

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the folds of mucous membrane surrounding the chain of ossicles, it is here that the abscess forms. Left to itself, the pus sooner or later breaks through the membrane and discharges into the meatus, with relief to all the symptoms, and in favourable cases with spontaneous cure in a few days. If, however, the bone has become infected, as when one of the ossicles is necrosed, or if, owing to carelessness or ignorance, a superadded infection from the meatus occurs, the discharge becomes chronic.

During the acute stage the disease may involve the mastoid cells, the lateral sinus, the labyrinth, the brain or the meninges.

Symptoms of acute catarrh.—In acute catarrh there are earache, deafness, and tinnitus, usually sudden in their onset, with a moderate amount of fever and malaise. After some hours or days, relief is afforded either by the spontaneous involution of the disease process or by rupture of the membrane and the discharge of the accumulated exudation. Recovery is usual, with restoration of normal hearing. Sometimes, however, the deafness persists and becomes chronic, or infection takes place through the perforation, and transforms the catarrhal into a purulent inflammation. The appearance of the membrane varies with the stage and severity of the disease, but as a rule the reddening is slight. Bulging, if present, affects the whole membrane, whereas in acute suppuration it is the postero-superior quadrant which is chiefly affected. This distinction, however, cannot always be made, so that before rupture takes place the only difference between catarrh and suppuration may lie in the comparative mildness of the local and general symptoms in the former condition.

Treatment of acute catarrh.—If seen early, the attack may be aborted by sending the patient to bed, giving a purge, and instilling into the ear drops consisting of glycer. acid. carbol. 2½ dr., glycer. to 1 oz., mixed. At the same time heat may be applied to the side of the head by means of a hot rubber bottle or fomentations.

If rupture of the membrane seems to be inevitable, this development should be forestalled and the membrane incised in the manner described in the next section (p. 441). If the symptoms appear to be moderating, this operation may be avoided, but in a doubtful case early incision should be practised. Antiseptically performed, it is a harmless procedure.

After the discharge has appeared, the dilute glycerin and carbolic-acid drops should be continued with the object of preventing further infection.

Symptoms of acute suppuration.—These are similar to those of acute catarrh, but, generally, much more severe. The earache is more intense, and the prostration greater. This is especially true of children, in whom an acute purulent otitis means a serious illness. In infants, in addition to high fever and restlessness, the appearance of convulsions, squint, and some head retraction may arouse fears of meningitis. In adults, the constitutional reaction is usually slight, even in acute suppuration. There may be no fever and no general disturbance. Even the pain may be trivial or absent, the only symptoms being dullness of hearing and the appearance of discharge.

In all cases of unexplained pyrexia in children it is important to make an examination of the tympanic membranes with light and speculum.

After from twenty-four hours to four days the pyrexia and pain are relieved by the bursting of the abscess through the membrane. If, twenty-four hours after this event, the pyrexia has not disappeared, there is still some pus retention, either in the tympanum itself, the perforation being too small, or in the mastoid cells or elsewhere. The relief to the pain is less immediate, and that symptom may continue for three or four days without any complication being present. In any event, a careful watch should be kept for the signs indicating mastoiditis, lateral-sinus thrombosis, labyrinthitis, etc., as long as the discharge continues.

Examination of the membrane before its rupture shows it to be livid-red, swollen, oedematous and bulging, with its landmarks altogether submerged. It is usually covered with a white pellicle—its necrosed but still adherent superficial layer, which is apt to be mistaken for a normal appearance, especially if the examination be too hastily made. In order to see the membrane, this pellicle must be carefully peeled off by means of a fine probe guarded with cotton-wool, a manoeuvre only possible in children if they are under an anæsthetic.

Shortly after perforation, the appearances are the same, with the addition of discharge, which at first is thin and bloodstained. Later it becomes purulent. In all save the mildest cases it is profuse.

In most cases recovery follows without treatment. In some, the preventable second infection takes place, and the discharge becomes chronic. In others, the necrosis of an ossicle or of some other osseous part leads to a continuance of the discharge in spite of the most careful meatal treatment. In a very small proportion of cases one of the complications supervenes.

Treatment of acute suppuration.—During the first twenty-four hours the treatment is the same as that for acute catarrh, with this addition, that a leech may be applied to the mastoid process. If these measures fail, and the bulging of the membrane shows the presence of pus, further temporizing is futile. The patient should be anesthetized (chloroform for a child and nitrous oxide for an adult), and a free incision made in the membrane. This little operation is well within the powers of the ordinary practitioner. It is easily performed; it is free from danger; and even if it is carried out before pus has formed no harm will follow. The *myringotome* is the most suitable instrument, but a tenotomy knife may be employed successfully in an emergency. After the meatus has been cleared of débris and thoroughly cleansed with mercury biniodide in spirit (1:500) a speculum is inserted, and under good illumination the point of the knife is plunged through the membrane close to the floor of the meatus and carried upwards to the roof in such a way as to incise the membrane from bottom to top. Blood flows freely through the opening, but it soon stops. Pus is seldom recognizable. A drain consisting of a few long strands of gauze is inserted through the speculum up to and, if possible, through the opening in the membrane, and is left there for twenty-four hours. A boric fomentation is then applied to the ear and secured with a pad of cotton-wool, and a bandage. In most cases the treatment is followed by immediate relief to the pyrexia and malaise. The pain disappears in a day or two, or even in less time. A few days later the discharge dries up, and the incision heals so perfectly that in a few weeks' time not even the scar is visible. In such cases the hearing is completely restored.

Incision should also be carried out after spontaneous rupture, if the temperature or the pain continue longer than the periods mentioned above.

If, however, in spite of free incision, the symptoms still persist, one or other of the

complications is present, and more extensive surgical measures are called for.

Sometimes, even after a free opening of the membrane, still more often after spontaneous rupture, the constitutional phenomena vanish while the discharge continues. In such cases, antiseptic drops (dilute glycerin of carbolic acid) should be employed, but if, in spite of careful meatal treatment, it has not ceased in three months a mastoid operation should be performed.

There is one warning necessary. If acute otitis be due to adenoids, these *should not be removed until the acute symptoms have completely subsided*.

2. CHRONIC SUPPURATION OF THE MIDDLE EAR

In tuberculosis of the middle ear, and in many cases of cholesteatoma, suppuration is chronic from the outset; otherwise it follows an acute otitis.

The distinction between acute and chronic suppuration lies chiefly in the fact that the latter is a mixed infection. Clinically, the term "chronic" is applied if the discharge has continued for three months or longer. A chronic suppuration may be rendered acute by an invasion of virulent organisms, and this acute exacerbation is responsible for many fatalities.

The disease is often associated with caries or necrosis of bone, and consequently with the formation of exuberant granulations or polypi. In the ear a polypus is merely an hypertrophied granulation, and is almost invariably due to suppuration. For this reason it generally recurs after removal, unless the disease is eradicated by a mastoid operation.

Symptoms.—There are discharge of pus, and more or less deafness and tinnitus. The disease is painless unless some complication is present. The membrane is seen to be more or less destroyed. Shrapnell's membrane is spared unless the ossicles have been destroyed and removed, when the whole *membrana tympani* may be absent. On the other hand, in what is termed *attic disease*, the only perforation present is through Shrapnell's membrane, and the vibrating membrane is intact, little or no loss of hearing resulting. This "perforation" is really the opening of a sinus which leads directly to some caries in the attic. A frequent cause is *cholesteatoma*. In this disease the middle-ear spaces become filled with disintegrated epidermal cells. As a consequence, fetid masses of pultaceous material

accumulate in the middle ear and in the meatus, and may open up the mastoid bone, erode their way into the labyrinth, or set up lateral-sinus thrombosis or some other complication. It is diagnosed by the presence of suppurative coupled with the existence in the meatus of a soft whitish material with a characteristic and most offensive odour.

Treatment.—The indications for immediate surgical interference (see p. 441) being absent, adenoids must be removed, and any nasal disease must receive attention. To ensure meatal cleanliness the patient should be taught to instil into the meatus twice daily a 10-volume-per-cent. solution of hydrogen peroxide. After the active foaming due to the peroxide has subsided, he should carefully mop the canal dry with sterile wool twisted securely round a sterile probe or bodkin. Finally, he should insert a few drops of an antiseptic solution and allow them to remain. The following are the antiseptic drops generally used :—

R^y Glycer. acid. carbol. ʒiiss.
Glycer. ad ʒi.

R^y Pulv. acid. bor. gr. xvi.
Aq. ad ʒi.

When granulations are present, alcohol should be added to the above formulæ, in quantities ranging from 50 per cent. upwards according to the patient's tolerance. Alcohol is the most efficient of all ear drops in suppurative, and the stronger the patient can bear it the better. If the discharge shows evidence of diminishing, meatal treatment may be continued so long as no sign of any complication appears. A strict watch should be kept for any symptoms of complication. A mastoid operation will be indicated if any of the following danger signals make their appearance : (1) Pain in the ear ; (2) frequent or persistent headache ; (3) vertigo even if slight, provided it is recurrent ; (4) pyrexia (99° F. or more) ; (5) obstinate granulations ; (6) polypus-formation ; (7) stenosis of the meatus ; (8) mastoid tenderness, œdema, and abscess ; (9) facial paralysis ; (10) extensive cholesteatomatous disease ; (11) persistent discharge in spite of careful meatal treatment, especially in patients whose life is passed beyond the reach of prompt surgical aid.

People with discharging ears should not plug the meatus with cotton-wool, nor should they be allowed to indulge in swimming, diving, etc.

Residual cicatrization.—In this condition, dry

perforations, scarring, etc., of the membrane show that the patient has passed through an attack of suppurative of the ear. Relief is sought for deafness. Some of the patients are the subjects of still recurring attacks of suppurative ; others may be unaware of ever having had any discharge. In the former class the hearing is usually more acute when the ear is discharging than when it is dry, probably because of the loosening of fibrous bands and the lubrication of the ossicular articulations by the moisture.

The treatment of the deafness is unsatisfactory, and, as a rule, little or nothing can be done. At times some benefit may be obtained by using an artificial tympanic membrane consisting of cotton-wool soaked in liquid paraffin and changed daily, but this device must be used cautiously or it will induce a renewal of suppurative. "Artificial drums" are generally useless and always dangerous. Sometimes we may improve the hearing by prescribing dilute glycerin and carbolic-acid drops (see above). Operative treatment is seldom advisable, and fibrolysin and its congeners have proved to be useless. Otomassage is sometimes employed in this condition.

3. CHRONIC CATARRH OF THE MIDDLE EAR

Two varieties are described—(a) chronic hypertrophic catarrh, and (b) chronic atrophic catarrh.

(a) **Chronic hypertrophic catarrh** is usually a sequel of chronic nasal and nasopharyngeal catarrh and, especially, of adenoids. It is characterized by œdema and hypertrophy of the mucous membrane of the middle ear and by a tendency to the exudation of sero-mucous fluid in the middle ear and tubal spaces.

The **symptoms** are tinnitus, deafness, and auditory hyperæsthesia, showing considerable variation in severity from day to day. The deafness is obstructive in type (see p. 440), and the membrane, on inspection, appears opaque and dull in hue, and often very much retracted.

Treatment consists in removing the nasal and naso-pharyngeal disease. Adenoids should be extirpated, and the nasal passages cleared and rendered healthy. It is particularly in this kind of deafness that benefit has followed the treatment of catarrhal changes about the Eustachian orifice, in the diagnosis of which the naso-pharyngoscope has proved of great value.

The ear should then be treated by repeated

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politzerization, catheterization (*see* EAR, EXAMINATION OF), and inflation into the middle ear through the Eustachian catheter of—

R̄ Menthol gr. iii.
Paraff. liq. ad ʒi.

or—

R̄ Zinc. chlor. gr. iii.
Ol. anilin. q.s.
Paraff. liq. ad ʒi.

This local treatment should be carried out at intervals of a few days as long as the hearing shows any tendency to improve. When the ultimate pitch of improvement has been reached the treatment may be remitted for a time and resumed again some months later.

(b) **Chronic atrophic catarrh.**—This condition, which may either arise independently or follow upon the hypertrophic variety, is characterized by cirrhotic and adhesive processes in and about the ossicles, in such wise as seriously to interfere with their mobility. As a rule both ears are affected, but one is usually worse than the other. The earliest symptom is tinnitus. This leads to middle-ear deafness, usually of a progressive character, although its progress may be very slow. At a later stage the middle-ear deafness may be followed by labyrinth deafness. The membrane is usually thin and atrophied, and may be so translucent that the incus is visible through it. It is often much indrawn and adherent to the inner wall of the tympanum.

Treatment.—On inflation with the catheter or Politzer's bag, little or no improvement in the hearing is experienced, and treatment is seldom beneficial. The internal administration of potassium iodide may give a little relief. The so-called re-educative methods are disappointing, and the operative measures that have been tried have proved to be altogether vain. In severe cases the patient should be encouraged to obtain and to use an artificial aid to hearing, of which there are many useful varieties on the market.

DAN M'KENZIE.

OTOMYCOSIS (*see* OTITIS EXTERNA).

OTORRHOEA (*see* OTITIS MEDIA).

OTOSCLEROSIS.—A condition in which deafness is due to bony ankylosis of the foot-plate of the stapes in the fenestra ovalis.

Etiology.—The disease is often hereditary,

and, although the most striking and pathetic cases are those which occur in young women, it may begin at any age. It is frequently found in old people. For some unknown reason, certain areas of the hard smooth bone of the labyrinth wall, particularly that around the oval window, become transformed into rough cancellous bone, relatively great in bulk. Since these outgrowths encroach upon the stapedio-vestibular articulation, they impede the movements of the stapes more and more until that ossicle becomes immobilized.

Symptoms.—Naturally, this process is productive of deafness of a gradually increasing character, at first obstructive in type (*see* p. 440), but, later, showing evidence of cochlear involvement, which may progress until the patient loses all perception of sound. In many cases, however, the deafness advances to a certain stage and then remains stationary. Debilitating illnesses, and especially parturition, always increase it.

Tinnitus is a pronounced and constant symptom, and in the great majority of cases makes its appearance before any defect in the hearing has been observed.

Diagnosis.—Identical symptoms are produced by those diseases of the middle ear which lead to a fixation of the stapes by fibrous or bony ankylosis, but in such cases the changes in the mucous membrane of the middle ear are plainly indicated when the membrana tympani is inspected. In true otosclerosis, on the other hand, the membrana tympani presents a normal appearance, and it may be found that the handle of the malleus is movable.

Treatment.—The ordinary methods of treating middle-ear deafness, by inflation with Politzer's bag and by the catheter, are useless or worse than useless in otosclerosis. The only local measure which is at all likely to confer even temporary benefit is otomassage by means of a rapidly operating air-pump. Internally, potassium or sodium iodide, in small doses over prolonged periods, has been advocated and should be tried. Operations of any kind should be avoided, a warning which is particularly applicable to operations on the throat, the nose, and the ear. Artificial aids to hearing should be employed as soon as the deafness is severe enough to interfere with the patient's hearing of conversation even when it is carried on in a moderately loud tone.

DAN M'KENZIE.

OVARIAN CYSTS

OVARIAN CYSTS.—A thoroughly satisfactory and inclusive **classification** of ovarian cysts is difficult to attain. For practical purposes we may divide them as follows:—

1. Simple follicular cysts.
2. Cysts of the corpus luteum.
3. Multilocular adenomatous cysts.
4. Papillomatous cysts.
5. Tubo-ovarian cysts.
6. Blood-cysts of the ovary, or hæmatomata.
7. Cystic teratomata, formerly called ovarian "dermoids."
8. Broad-ligament cysts.

1. **Simple follicular cysts.**—These are formed from distended Graafian follicles, probably aborted follicles from which the ovum has disappeared without the follicle undergoing maturation. They seldom grow larger than a Tangerine orange, are always unilocular, and often multiple. They rarely form adhesions. A slight degree of dilatation of follicles is common, and it is difficult to say where the line should be drawn between what can be considered normal and what is pathological. Many women whose ovaries contain numerous distended follicles complain of no symptoms whatever. Small follicular cysts project from the surface of the ovary, while, if a cyst is comparatively large, the ovary seems to be attached to the surface of the cyst. It is possible that a cyst, the size of a Tangerine orange, is made up of two follicles whose contiguous walls have atrophied. The contents are a clear yellow watery fluid; the wall is thin, and, if the cyst is small, is lined by epithelium from the *membrana granulosa*. In the larger cysts the epithelium may atrophy from pressure. If a cyst is found to be of this nature at operation it should be excised and the practically normal ovary left behind.

It is not very uncommon for such a cyst to disappear during bimanual examination, its thin wall having been ruptured by pressure. No harm results from the flow of the contents into the peritoneal cavity.

2. **Cysts of the corpus luteum.**—These are not very common, and seldom grow to a size larger than a golf-ball. The wall is thick, lined by a layer of lutein cells, which are ordinarily convoluted in small cysts, but become stretched out into a smooth lining in the larger tumours. They are sometimes found in nulliparæ. They contain a clear fluid, and are of no importance clinically unless they suppurate. In my experience a considerable

collection of pus in an ovary removed with a pyosalpinx has frequently proved to be contained in a corpus luteum cyst.

A peculiar variety of tumour is found in many cases, if not in all, in which the uterus contains a vesicular mole. The tumour is seldom much larger than a fetal head, usually a good deal smaller than this. From its surface project many thin-walled glistening cysts about the size of grapes. On section the ovarian stroma is found to be full of lutein cells. After expulsion of the mole the ovaries gradually return to their normal size. The walls of these cysts are sometimes so thin, being described as looking like small soap-bubbles, that they may be easily ruptured by pressure, or by increase of tension after torsion of the pedicle.

3. **Multilocular adenomatous cysts.**—These, the commonest large ovarian cysts, are also called cyst-adenomata, or glandular cysts. They may be divided into two classes: (a) the common pseudo-mucinous cysts and (b) the much less common serous adenomata.

(a) **Pseudo-mucinous cysts.**—These, the common multilocular cysts, grow rapidly and may reach to an enormous size, growing until they kill the patient. The surface is of a shining pearly grey or white colour, and often irregular from the bulging of small cysts from the main swelling. In many cases there is one large main loculus, the interior of which shows the remains of party walls. These tumours frequently contain much tissue which is apparently solid, but is seen on section to have a sort of honeycomb structure. As a rule, the main walls are fairly tough, containing a good deal of fibrous tissue, but in some cases the walls, apart from degeneration, are thin and easily torn. The contents are not serous but mucoid or gummy. They are often too thick to run through even a large cannula, and look like ropy mucus, or they may resemble pus, from the presence of shed cells; sometimes they form an almost solid jelly. They may be colourless, yellow or green, and there are often definite traces of blood in some of the loculi. The contents of the loculi of one cyst may vary greatly as regards colour and consistence. The cavities are lined by glandular epithelium arranged in a single layer, though there is often proliferation with formation of microscopic tufts projecting into the glandular spaces. The epithelium consists of high columnar cells, some of which are swollen with pseudo-mucin, while goblet cells are common. As a rule, there is no difficulty in deciding that the macroscopic appearances denote that the cyst is

innocent. In a small number of cases where there is solid tissue in the wall of the cyst, naked-eye examination may suggest the presence of carcinoma. In these circumstances microscopical sections should be prepared from the solid tissue to settle the question of malignancy. A malignant portion in an otherwise innocent tumour is more likely to be found in middle-aged or elderly patients than in young women. Occasionally one loculus in a multilocular adenomatous cyst may be "dermoid," containing sebaceous matter, hair, etc. Some of these tumours, as we have seen, have exceedingly thin walls, so thin that they may be described as a soft membrane rather than a tough wall containing fibrous tissue. They may give way, with escape of contents into the peritoneal cavity, without any recognizable cause, apparently as the result of tension during their growth.

(b) *Serous adenomata* may form large tumours. They have thin tough walls formed by fibrous tissue lined with columnar epithelium, and have fewer loculi than the pseudo-mucinous cysts. They do not grow so rapidly as the more common variety.

The etiology of the cyst-adenomata is unknown. Probably they are derived from downgrowths from the surface epithelium. They are more likely than any other ovarian tumour to kill by mere size. Usually they occur only during menstrual life. It is not uncommon for them to be bilateral. When large they are, as a rule, adherent over part of their surface, and there is often a small amount of free fluid present, but these tumours do not cause ascites in sufficient quantity to be recognized clinically unless some change has occurred, generally as the result of twisting of the pedicle.

After rupture, either spontaneous or during an operation for their removal, there may be "recurrences," the so-called pseudo-myxoma of the peritoneum, masses of jelly more or less circumscribed by a thin membrane being found on the peritoneum, sometimes under the peritoneum, between the folds of the mesentery and in the intestinal walls. This recurrence, which has nothing to do with carcinoma, seems to occur most frequently in the soft, thin-walled pseudo-mucinous cysts. In one case under my own care I opened the abdomen seven times, removing masses of jelly which were partly free in the abdominal cavity, partly surrounded by a more or less definite membrane, and partly invading the wall of intestine.

Apart from these "recurrences," rupture of a pseudo-mucinous cyst sometimes results in great distension of the abdomen with more or less fluid jelly which the peritoneum is unable to absorb, although no implantation occurs. When the abdomen is opened in such a case, the ruptured cyst removed with all the loose pseudo-mucin, and the peritoneal cavity flushed out with saline solution, no further accumulation may take place.

4. **Papillomatous tumours.**—It is not uncommon to find a few hard fibrous papillomata on the inner wall of a broad-ligament cyst, but the term "papillomatous ovarian tumour" is reserved for a different class of tumour. Two varieties are met with, one definitely cystic, the other made up almost entirely of papillomatous masses with a few small cystic cavities in its interior. The cystic variety is usually unilocular, containing a clear or cloudy thin fluid. On the inner surface are masses of papillomata, some of which may perforate the wall and appear on the outer surface. These tumours are seldom large, are frequently bilateral, and are often accompanied by ascites. They sometimes grow between the layers of the broad ligament.

When there are papillomata on the outside of the tumour, and in the rare cases in which the cyst ruptures, there is often dissemination of small papillomata all over the contents of the peritoneal cavity, sometimes closely resembling the appearance seen in tuberculous peritonitis. After removal of the ovarian tumour these secondary papillomata sometimes disappear. Papillomatous tumours are probably derived from proliferation of the germinal epithelium. The papillomata vary in microscopic structure. In most cases they are composed of a connective-tissue core covered with a single layer of low columnar or cubical epithelium. In other cases they are definitely carcinoma, with branched papillae and much proliferation of epithelium. Between the two extremes are formations in which there is some proliferation but nothing that can be definitely called malignant. The non-malignant character of some of these tumours, even when they cause ascites, was well shown in the case of a patient from whom bilateral papillomatous ovarian tumours were removed after she had been tapped thirteen times for ascites. Three years after the operation she was in good health, with no return of ascites.

5. **Tubo-ovarian cysts.**—For the formation of a tubo-ovarian cyst it is necessary that a di-

times impossible until the abdomen is opened, they are usually included among ovarian cysts. They used commonly to be grouped together under the heading of parovarian cysts, but it is generally considered now that most large broad-ligament cysts are developed from the ovarian fimbria.

Two different varieties of broad-ligament cysts must therefore be described: (i) the fimbrial cyst, (ii) the parovarian cyst. A third variety, very commonly found but seldom of any clinical importance, may be included, viz. the hydatid of Morgagni.

i. *Fimbrial cysts*.—These, as their name implies, arise from developmental relics in the ovarian fimbria. They are thin-walled cysts which grow to a very large size until they fill up the cavity of the pelvis and a considerable part of the abdominal cavity. When small, a fimbrial cyst is situated at the outer side of the mesosalpinx, and as it grows larger it separates the anterior and posterior layers of the broad ligament, acquiring a complete though often loose peritoneal investment, with the Fallopian tube stretched over it. The abdominal ostium of the tube becomes stretched in a characteristic manner, with the ovarian fimbria elongated and thrown into prominence. Sometimes the cyst enlarges entirely at the expense of the mesosalpinx, in which case the ovary is at the lower part of the cyst, and the deeper part of the broad ligament forms a pedicle. In other cases the cyst burrows into the deeper part of the broad ligament as well, until it reaches the pelvic floor, and might be called retroperitoneal. The ovary will then be lifted up as the cyst increases in size. Sometimes the ovary is very much flattened by pressure. This is the typical broad-ligament cyst which fills up the cavity of the pelvis to a large extent, pushing the uterus to one side, and causing difficulty in micturition or even retention of urine. The former variety may undergo axial rotation, the latter cannot, as it has no pedicle.

These cysts are thin-walled, unilocular, and contain a clear, colourless, watery fluid. Not uncommonly there are a few fibrous papillomata on their inner surface.

ii. *Parovarian cysts* grow from the parovarium or organ of Rosenmüller, which is situated between the layers of the mesosalpinx. They are necessarily surrounded by peritoneum, with the Fallopian tube above them and the ovary below and behind. They seldom grow to a larger size than that of an ordinary

orange, and frequently undergo twisting of the pedicle.

iii. Hanging from the fimbriated extremity is usually seen a small pedunculated cyst, the *hydatid of Morgagni*, commonly the size of a pea. It may be a good deal larger than this, but is of no clinical importance unless its pedicle become twisted—a rare occurrence.

Pseudo-broad-ligament cysts.—It is not uncommon for a cyst-like swelling of moderate size, especially an ovarian abscess due to puerperal sepsis, to grow forwards and upwards in such a manner that the broad ligament forms a hood over it. Superficial examination of the relations of the tumour may lead to the erroneous idea that it is situated between the layers of the broad ligament, but a careful examination will show that it can be shelled out more or less completely from the bed which it has made for itself without opening the broad ligament.

Symptoms of ovarian cysts.—Many ovarian cysts, even when of large size, cause no symptoms whatever. Even enlargement of the abdomen is often found out accidentally, sometimes by the dressmaker.

Effect on menstruation.—There is a belief widely spread among medical men that ovarian tumours cause amenorrhœa. This is incorrect, amenorrhœa due to ovarian tumours being one of the rarest conditions found in gynecology. A so-called ovarian tumour, supposed to be producing amenorrhœa, is in almost every case found to be a pregnant uterus. Very rarely amenorrhœa may be caused by bilateral malignant ovarian tumours. It is not unusual for a patient with an ovarian tumour to complain of excessive bleeding at the catamenia, and uterine hæmorrhage occurring many years after the menopause occasionally accompanies the growth of an ovarian tumour. In the majority of cases, however, ovarian tumours exert no influence on menstruation.

Pain.—Most ovarian cysts cause no pain. When pain is felt it is often due to the presence of adhesions, but it cannot always be accounted for in this way, as it may be complained of when the tumour is small and has no adhesions. Possibly the pain in some of these cases may be due to tension in the cyst. Ovarian tumours may cause symptoms by pressure in the pelvis or in the abdomen, by adhesion to sensitive structures, and—the most acute symptoms—by torsion of the pedicle and its sequelæ.

Pressure in the pelvis.—An ovarian tumour is

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less likely to become impacted in the pelvis than a uterine tumour, though occasionally this does occur, with or without the presence of adhesions. If an ovarian cyst remains in the pelvis instead of rising up into the abdominal cavity, after it has reached the size of a foetal head, it will cause discomfort and pressure symptoms. The cause of its remaining in the pelvis may be the presence of adhesions, the fact that the cyst is situated in the deep part of the broad ligament, or simply that it fits the pelvic cavity so accurately that a good deal of force is required to lift it out and up into the abdomen. The last condition is rare, but occasionally the help of an assistant is necessary during the performance of ovariectomy to push up through the posterior fornix so as to dislodge a cyst which cannot be pulled up from above. An inflamed ovarian "dermoid," or cystic teratoma, is the variety of ovarian cyst which most commonly causes pressure symptoms in the pelvis. A broad-ligament tumour, even when growing in the deep part of the broad ligament, is usually able to extend upwards though its base is still attached low down. It is not very uncommon to find, in the case of a large ovarian cyst which occupies a considerable part of the abdomen, that the pelvis is filled up tightly by a cystic swelling. Operation often shows that there are two tumours, one in the abdomen and the other, the smaller, in the pelvis. If this is not the case it will be found that one loculus, projecting from the surface of the large cyst, is occupying the pelvic cavity.

The most common and most important pressure symptom in the pelvis is retention of urine from displacement upwards of the cervix uteri, with consequent elongation of the urethra, or from pressure of the urethra against the symphysis pubis. This retention may lead to overflow with danger of cystitis, and subsequent ascending pyelonephritis. There may be a feeling of discomfort in the rectum, as if there were a faecal mass to be passed, and some difficulty in defaecation, but nothing that can be called intestinal obstruction. If the patient suffers from haemorrhoids, trouble from these is likely to be accentuated. Pain from pressure on the nerves, and oedema of thighs and legs from pressure on the pelvic veins, are seldom, if ever, met with except in the case of malignant tumours, though oedema is very commonly produced by large innocent tumours.

Pressure symptoms in the abdomen.—Some patients complain of discomfort with a com-

paratively small ovarian cyst, while others notice nothing except a "fullness of the stomach," although the cyst may be as large as the pregnant uterus at seven or eight months or more. If the cyst grows to a very large size before its removal—a comparatively rare occurrence at the present day, when abdominal surgeons are so numerous—pressure symptoms in the abdomen may cause great distress. The typical "ovarian facies" is seldom seen now—the pinched face, wasted chest and arms, enormous abdomen and oedematous legs—but there are still many women met with who have large ovarian cysts. There may be (a) pressure on the diaphragm, causing palpitation, dyspnoea and a feeling of faintness on exertion, and orthopnoea, the patient finding that she cannot sleep lying down, but has to be propped up; (b) pressure on the stomach, causing dyspepsia and discomfort; (c) pressure on veins and lymphatics, causing oedema of the abdominal wall, thighs and legs; (d) pressure on the bladder, causing frequency of micturition, and sometimes involuntary escape of a few drops of urine on straining, coughing, etc.

With these very large ovarian cysts there is often a small amount of albumin in the urine, which disappears after the operation.

Adhesions.—As a general rule, to which there are many exceptions, small ovarian cysts are free from adhesions, while large ones are usually adherent over some part of their surface. Extensive adhesions may be found during ovariectomy in a case where there had been no complaint of pain, but frequently pain and even tenderness over some part of the tumour may be caused by adhesions, chiefly to small intestine; adhesions to omentum and to the parietal peritoneum do not necessarily cause any symptoms. After torsion of the pedicle, adhesions are the rule if operation is delayed for many days, and with a suppurating ovarian cyst or a cyst in connexion with pyosalpinx, adhesions may be universal, making ovariectomy a difficult operation. If any adhesions are present the great omentum is almost certain to be involved. The anterior abdominal wall, intestine, uterus, broad ligament, and the peritoneum of the pelvic floor probably come next in frequency, while the bladder is adherent least often, and usually only in cases where the tumour lies in front of the uterus. Adhesions to small intestine and bladder are those which cause the most trouble in separation.

Ovarian tumours with pregnancy and labour.—Ovarian tumours may cause trouble in labour,

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especially if they are comparatively small, by obstructing the passage of the child through the pelvis. Pregnancy is often complicated or accompanied by the presence of an ovarian cyst; this provides one of the reasons why routine examination in the course of every pregnancy is to be recommended. Ovariectomy during the first half of pregnancy does not often cause abortion, but in the later months of pregnancy there is some danger of premature labour following the operation. For this reason some operators, who would not hesitate to advise removal of an ovarian tumour during the first half of pregnancy, would prefer, if the patient were first seen about the twenty-fifth week, to keep her under observation, and to postpone operation until there was a good chance of the child surviving if labour followed soon after the operation.

Ovarian tumours seldom cause *prolapse of the uterus and vaginal walls*, but sometimes increase the degree of an already existing prolapse.

Accidents and secondary changes. *Torsion of the pedicle.*—Ovarian tumours possess a pedicle, unless their position is between the layers of the broad ligament, or they have grown up from behind and wrapped the broad ligament over themselves like a hood. This pedicle consists, on the outer side, of the ovario-pelvic or infundibulo-pelvic ligament, in which are the ovarian artery and veins; and, on the uterine side, of the Fallopian tube, ovarian ligament, with the upper part of the broad ligament and the ovarian artery and veins; while the middle portion of the pedicle is made up of the broad ligament. In length and breadth ovarian pedicles vary greatly. Sometimes the pedicle is narrow, and so long that the tumour has a very wide range of movement. In other cases it is broad, and so short that the movements of the tumour are much more restricted. The longer and narrower the pedicle, the more liable is torsion to occur, provided, of course, that the cyst is not adherent to surrounding structures. By torsion of the pedicle is meant a revolution of the tumour on a vertical axis so that the pedicle becomes twisted on itself. It is unusual to find a very large tumour undergoing torsion, but torsion of tumours up to the size of coco-nuts is common.

This accident, which is especially common during pregnancy, labour, and the puerperium, is one of the most frequent causes of acute

abdominal symptoms in women. Any variety of ovarian tumour may undergo axial rotation if it has a distinct pedicle. If the pedicle is very long and cordlike it is possible for torsion to produce no symptoms—at the operation one or one-and-a-half twists may be found with no resulting change in the cyst; in other cases less than a complete twist may cause deep congestion of the tumour. Sometimes there is an evident cause, such as a sudden twist of the body—e.g. leaning out of bed to pick up something from the floor; but in most cases no definite cause can be found for the rotation of the tumour. When rotation occurs during pregnancy, labour, or the puerperium, it can be explained by the gradual or sudden change in the size or position of the uterus, necessitating some movement of the tumour. In other cases the shape of the tumour, one part of it projecting acutely from the general surface, may explain the change in its position. It is said that the rotation is more commonly in the direction of the movement of the hands of a clock. This is of little more than academic interest, except that it suggests that the alternate filling and emptying of the pelvic colon may be a cause of the rotation.

In a typical case the patient suddenly experiences severe pain, and may faint or collapse. The pain continues, and is accompanied by vomiting, and by gradually increasing distension of the abdomen from partial paralysis of intestine. If the presence of the tumour has been recognized before, it may be noticed that it feels more tense, and is enlarged and tender. In rare cases the symptoms may all subside, and at a subsequent operation no twist be found; probably the cyst rotated back in the opposite direction and the twist became undone. In torsion with acute symptoms, diagnosis must be made from perforation of a gastric or duodenal ulcer, or of the appendix, from acute strangulation of intestine in a hernial sac or within the abdominal cavity, from biliary or renal colic, and, most commonly, from acute intraperitoneal hæmorrhage from a pregnant Fallopian tube. In the large majority of cases the history, with careful abdominal and bimanual examination, makes the diagnosis clear. It is rare for an ovarian tumour too small to be found by bimanual examination to cause acute symptoms by torsion of its pedicle, though I have recorded a case in which torsion of the pedicle of a hydatid of Morgagni the size of a cherry produced acute symptoms. In such a case a

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certain diagnosis cannot be made without abdominal section, although the real cause may be suspected. In some cases there is difficulty in diagnosis between acute torsion of the pedicle of an ovarian tumour and fresh bleeding in a case of tubal pregnancy where an encysted hæmatocele has been formed before, and very rarely the differential diagnosis between these two conditions may be practically impossible. It must be remembered that although acute torsion of the pedicle of an ovarian cyst may produce physical signs and symptoms closely resembling those of intraperitoneal hæmorrhage, it will not cause anæmia, except in the rare cases where bleeding occurs as the result of the torsion. As a rule, a twisted ovarian tumour can be felt as a tense, tender, movable swelling, and, very rarely, if the abdominal wall is thin the twisted pedicle itself may be felt on bimanual examination. Red degeneration of a uterine fibroid is not uncommonly mistaken for torsion of the pedicle of an ovarian tumour without the most acute symptoms.

The first effect of acute torsion is to cause venous engorgement of the tumour, the thin walls of the veins being more effectually compressed than the thicker walls of the arteries. There is usually some bleeding into the tissues of the cyst-wall, comparatively rarely bleeding into the cavity of the cyst, and more rarely, and only in the case of soft-walled tumours, e.g. the polycystic lutein tumour which often accompanies hydatidiform mole, the cyst-wall may give way and bleeding occur into the peritoneal cavity. Occasionally a vein on the external surface of the tumour may rupture and cause intraperitoneal hæmorrhage. I have seen this only with acute torsion of the pedicle of a large ovarian fibroid, never with a cyst. If the patient is operated on soon after the torsion takes place, the tumour is found to be of a dark plum colour or almost black, the appearance resembling that of intestine in a strangulated hernia. On section the tissues are seen to be full of blood, resembling the tissue of the spleen. If the patient is not operated on, the acute symptoms, as a rule, pass off gradually, but she is left with discomfort, a certain amount of pain, and more or less evidence of toxæmia. Numerous, often universal, adhesions are left as the result of localized peritonitis, and there may be large flakes of lymph on the surface of the tumour. The longer the interval between the twisting and the operation, the more dense do the adhesions become, and the more likelihood is there

of the cyst becoming infected. If an operation is performed some weeks after the twisting, the tumour may contain pus or may be dead-looking, of a dirty yellow colour, with soft friable walls, which are likely to give way during operation, however gentle the operator may be. A cyst which is not removed until several weeks or months after the twist occurred may be found to be flabby, i.e. not so tightly distended as is usual with ovarian cysts, and there may be some calcareous degeneration of its wall.

In rare cases a thin pedicle which is tightly twisted may give way, so that the cyst loses all connexion with the broad ligament, and remains attached to great omentum or some abdominal viscus, its nutrition being carried on by means of adhesions. This happens most commonly in the case of cystic teratomata.

In those rare instances where the cyst has acquired adhesions to intestine before undergoing axial rotation, intestinal obstruction may be caused when the rotation occurs.

As will be seen later, axial rotation of an ovarian tumour is the commonest cause of bleeding into the cavity of the cyst, of suppuration, and of gangrene.

Suppuration.—Abscess of the ovary, i.e. the presence of one or several cavities filled with pus, is a common accompaniment of a pyosalpinx, especially if puerperal in origin. Apart from this, suppuration of an ovarian cyst is most often preceded by torsion of the pedicle or by bruising in labour. In some cases, however, no cause can be found for the suppuration, unless the numerous adhesions to intestine which are usually present in such cases can be taken to be the cause of the infection. Suppuration of an ovarian cyst from any cause is rare; it is less rare in "dermoids" than in other varieties of cyst.

Sometimes there are no symptoms to indicate that the contents of the cyst have been infected, i.e. there is no absorption of toxins; but as a rule pyrexia, ill-health, abdominal pain, and discomfort, considered together with the facts that the cyst is more or less fixed and that attempts to move it cause pain, suggest the probable diagnosis. In many cases the evidence of toxæmia is striking. Rupture of a cyst due to suppuration cannot be considered common, but in a small proportion of cases the weakest part of the cyst-wall may give way, with escape of the contents, most commonly into the large intestine, especially the rectum, seldom into the general peritoneal

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cavity, as this is shut off by omental and intestinal adhesions. Occasionally the contents of a suppurating ovarian cyst, if no operation is performed, may be discharged through the anterior abdominal wall, but the only common exit for the pus besides the large intestine is the bladder. The vaginal wall is so tough that discharge of the pus by this route is most unlikely. Ovarian "dermoids" (cystic teratomata) are the cysts most likely to open into the bladder, because, more frequently than other varieties of ovarian cysts, they lie in front of the uterus. This variety of cyst is also the one which most commonly becomes infected—it grows slowly and remains long in the pelvis, and consequently runs a greater risk than other varieties of being crushed by the foetal head in labour. Gebhard suggested that the frequent suppuration of these teratomata may be accounted for by the fact that the distal ends of the hairs sometimes protrude through the wall of the cyst and irritate intestine. I have seen the ends of hairs projecting through the cyst-wall into the peritoneal cavity.

Bleeding into ovarian cysts.—Bleeding into ovarian cysts of sufficient severity to have any clinical significance is a rare occurrence. The commonest cause of it is acute torsion of the pedicle, in rare cases of which the increase of tension may be sufficient to rupture the wall of the cyst, when bleeding occurs into the peritoneal cavity. A slight amount of bleeding, of no clinical importance, may be caused by breaking down of party walls in the growth of a multilocular cyst.

Rupture of ovarian cysts.—If the "pointing" of infected ovarian cysts is excluded, rupture must be looked on as a rare accident. As stated above, soft-walled cysts may occasionally give way after acute torsion of the pedicle. Trauma, such as a violent blow on the abdomen, or severe pressure as in being run over, may cause rupture of a thin-walled cyst, and minute ovarian cysts are sometimes ruptured on bimanual examination. Apart from these causes, the adenomatous cysts, which are described above as being of the thin-walled variety, are the most likely to rupture without any obvious cause. In some cases the patient may experience no symptoms, and may exhibit no physical signs indicative of the rupture of the cyst, the absorptive power of the peritoneum being able to cope with the activity of the cyst-wall. In most cases, however, the patient suffers a certain amount of pain and shock when the cyst ruptures, and may note

that the tumour has become smaller or has disappeared, and there is evidence of free fluid in the abdomen. Many of the pseudo-mucinous cysts secrete a jelly which the peritoneum cannot absorb (see p. 445). The abdomen may contain a large quantity of this substance more or less walled in by adhesions and a pseudo-capsule. If a cystic teratoma, not suppurating, ruptures into the peritoneal cavity, a severe degree of irritation, leading to localized peritonitis, may be caused. Sometimes a peculiar epithelial infection occurs, numerous secondary tumours being found in the abdomen, usually small.

Gangrene of ovarian cysts.—This is fortunately a rare occurrence. It may follow an unusually acute torsion of the pedicle, though torsion that might be sufficiently acute to cause gangrene brings about symptoms of such severity as to lead, in most cases, to operation before gangrene occurs. Apart from torsion, gangrene may follow infection of such severity as to kill the tissues of the cyst. A diagnosis cannot be made until the abdomen is opened, the symptoms being those of a severe toxæmia, which will probably be explained as being due to suppuration.

Malignant growth.—There is no doubt that malignant growth in ovarian cysts is a comparatively common occurrence. The papillomatous and multilocular adenomatous cysts are the tumours in which carcinomatous growth is most likely to be found. Carcinoma is met with also in teratomatous tumours. In many cases in which carcinoma occurs in a cyst there are no symptoms suggestive of the presence of malignant disease, and a casual naked-eye examination of the tumour after removal may not necessarily arouse suspicion, though microscopic examination leaves no doubt that part of the tumour must be called carcinoma.

The occurrence of carcinoma in what seems clinically an innocent tumour is one of the reasons for advising removal of any ovarian tumour as soon as possible.

Sarcoma is found, less commonly than carcinoma, in the teratomatous tumours and in adenomatous and papillomatous cysts.

Diagnosis.—In considering the diagnosis of ovarian cysts, it is convenient to divide them into two classes—(1) those situated chiefly in the pelvis, that is, not large enough to be abdominal tumours; (2) those chiefly in the abdomen, when they can be felt by abdominal examination. In the diagnosis of medium-sized and large cysts the most common mis-

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takes in diagnosis are made between ovarian cysts, the full bladder, the pregnant uterus, uterine tumours and ascites. There are many kinds of swellings which may be confused with small ovarian cysts.

(1) **Diagnosis of a small ovarian cyst.**—It is essential for the diagnosis of a small intrapelvic tumour that the bladder, rectum, and sigmoid flexure should be emptied. Free mobility and absence of tenderness are generally characteristic features of a small ovarian tumour. To distinguish such a tumour from an *enlarged uterus* requires a careful bimanual examination, especially if the uterus is bent backwards or to one side, and in the second and third months of pregnancy, when the uterus is globular and elastic and Hegar's sign is present, i.e. there is a marked softening of the lower uterine segment. If the uterus is retroverted the body should be pushed up if possible, when the diagnosis becomes easier. If the uterus is irregularly enlarged with a subperitoneal fibroid projecting from its surface, the fact that the swelling is definitely one with the uterus, and that it moves with the cervix—i.e. that when the swelling is displaced the cervix moves, and when the cervix is displaced the swelling can be felt to move—distinguishes a uterine from an ovarian tumour. With a *pedunculated subperitoneal fibroid* there may be more difficulty; the hardness of the tumour and its more intimate connexion with the uterus, together with the fact that if there is a pedunculated subperitoneal fibroid the uterus is often irregularly enlarged by other fibroids, are the chief points in the differential diagnosis. From *inflammatory swellings*, pyo-salpinx or salpingo-oöphoritis without the presence of pus, an ovarian cyst is usually distinguished by the fact that the inflammatory swelling is tender and more or less fixed, while the history will often help. It may be impossible to distinguish a small inflamed suppurating ovarian cyst from a pyo-salpinx. A tumour which feels like a small ovarian cyst and is fairly movable, in spite of being tender, is likely to be a hydro-salpinx. If bimanual examination is easy it may be possible to distinguish the retort-shape of a hydro-salpinx.

Faecal masses, which may be present in the rectum or the lower part of the sigmoid flexure in spite of the patient having frequent motions, are sometimes mistaken for ovarian tumours. As a rule, however, they can be felt by rectal examination, and if they are too high up for this, the fact that they have no evident con-

nexion with the uterus, and that they can be permanently indented by pressure with the finger-tip, ought to serve to distinguish them from tumours of the ovary.

Mistakes are sometimes made in diagnosis between *pregnant tubes* and small ovarian cysts. The history, the physical signs, and the symptoms ought, as a rule, to prevent the mistake being made, as in the case of tubal pregnancy there is generally a history of one menstrual period being missed, a slight amount of irregular hemorrhage, sometimes with passage of a decidua cast, and pain, while on bimanual examination the uterus is found to be enlarged. A small *hæmatocele* also may cause difficulty in diagnosis unless the history is carefully considered.

In exceptional cases the bladder-wall may be so much thickened by chronic cystitis that the *empty bladder* is thought to be a tumour in the utero-vesical pouch.

Carcinoma or diverticulitis of the pelvic colon with surrounding inflammation is likely to be mistaken for a small fixed left-sided ovarian tumour. A fixed painful swelling in the left iliac fossa of a middle-aged or elderly woman should always be looked on with suspicion, and a thorough examination, including rectal examination, should be made under anæsthesia before an opinion is given.

In cases of doubt an examination under an anæsthetic may make the diagnosis certain. It is only rarely that both ovaries can be identified when there is a swelling of some other organ in the pelvis.

(2) **Diagnosis of larger ovarian cysts.**—Ovarian cysts large enough to be felt on bimanual examination may be confused with a large number of conditions, of which the full bladder, the pregnant uterus, ascites, and "phantom" tumours are the most common. It must be remembered that in many cases a large ovarian tumour cannot be felt on vaginal examination. If a large tumour is globular it sits on the brim of the pelvis rather than dips into it. In many cases, however, a projection on the surface of a large tumour dips down into the pelvis. Routine examination is the most important safeguard against mistakes in diagnosis, and a catheter must be passed in every case where there is possibility of doubt. Unless this is done a *full bladder* will often be called an ovarian cyst. A normal *pregnant uterus* is very frequently diagnosed as an ovarian cyst, usually in the first pregnancy in an unmarried woman. The erroneous idea that ovarian

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cysts cause amenorrhœa is partly responsible for the mistake, but neglect to make a routine examination is still more responsible. Careful palpation and "dipping" will usually show that there is a fetus in the tumour. Auscultation will reveal the presence of foetal heart-beats, examination of the breasts will show activity, and pigmentation and skin-cracks will usually be seen in the abdominal wall. On vaginal examination the bluish coloration of the vaginal walls and cervix, the softness of the cervix, the expansion of the lower uterine segment, and the fact that the uterus cannot be separated from the tumour, will all suggest the correct diagnosis. If hydramnion is present, diagnosis may be more difficult, but routine examination will nearly always show that the patient is pregnant, and the question to be settled will be, Is there pregnancy with an ovarian cyst, or is there hydramnion? The range of mobility of an ovarian cyst is usually much greater than that of a pregnant uterus. If a tumour, the nature of which is doubtful, can be displaced upwards so much that the hand can dip in between it and the symphysis pubis, it is much more likely to be an ovarian tumour than a pregnant uterus. It must be remembered that the pregnant uterus is the most common swelling in the female abdomen, and the safest way to tackle the diagnosis of an abdominal tumour is to start by proving or excluding pregnancy.

The differential diagnosis between *ascites* and ovarian cyst is usually, but not always, easy. Percussion is one of the most important factors in the diagnosis. With *ascites* there is, as a rule, resonance over the front of the abdomen with dullness in both flanks. When the patient lies on either side the upper flank is resonant while the lower one is dull. If the abdomen contains a large ovarian cyst there is dullness over the front of the abdomen with resonance in both flanks, whether the patient lies on her back or side. If the abdomen is tightly distended with free fluid its shape may resemble that of an abdomen containing a large ovarian cyst, but if the fluid is not under great tension it tends to sag on both sides when the patient is lying on her back, and is less prominent in front. In cases of *ascites* due to tubercle or carcinoma, in which the mesentery is thickened or contracted, the intestine may be unable to float up to the surface, so that percussion is a less trustworthy guide, and the thickening of the parietal peritoneum in such cases may suggest strongly that there is

a cyst-wall below the abdominal wall. Vaginal examination may be of value in such a case by revealing the presence of nodular masses in the pelvis, but bimanual examination is of little use. In some cases with an ovarian cyst, and in most cases with a solid ovarian tumour, there is *ascites* present. Administration of an anæsthetic may be necessary before the presence of a tumour with free fluid can be definitely made out.

Uterine fibroids, particularly large pedunculated subperitoneal tumours, may be mistaken for ovarian tumours. There is usually a good deal of difference in the shape of the abdomen. A solid uterine fibroid generally stands out prominently, so that when the patient is lying on her back the abdominal wall above the tumour falls away more or less sharply, while a cystic swelling is more moulded by the abdominal wall, so that the contour of the abdomen is more rounded and regular. In rare cases the round ligaments can be felt by abdominal examination, proving that the tumour is uterine, but it is difficult to be certain that what is felt is the round ligament. As a general rule, ovarian tumours are cystic and uterine fibroids are solid, and in most cases fibroids have a more intimate connexion with the uterus than have ovarian tumours, so that on bimanual examination when the tumour is moved the cervix moves, and when the cervix is moved the tumour moves. If, however, a pedunculated subperitoneal fibroid has become cystic, diagnosis is impossible until the abdomen has been opened. If the uterus is generally enlarged by fibroids, bimanual examination usually shows that the tumour is uterine—the body of the uterus cannot be felt apart from it. A tumour which is growing in the broad ligament, or an ovarian tumour which is adherent to the uterus, may produce physical signs which are practically indistinguishable from those produced by uterine fibroids. Examination by the uterine sound gives little help; the uterine cavity is more likely to be elongated by a uterine than by an ovarian tumour, but a large uterine fibroid may be present without any elongation of the uterine cavity, while on the other hand the uterine cavity may be elongated by ovarian tumours which are adherent to the uterus or by broad-ligament tumours, particularly if they are bilateral.

Enlargement of the abdomen due to the presence of *much fat in the abdominal wall and omentum* may be thought to be an ovarian tumour, and the patient herself may be com-

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vinced that she has a tumour. Percussion is likely to produce a dull or partially-dull note, especially over the lower part of the abdomen. If the patient can be got to relax her abdominal muscles, the hands can be gradually sunk to such a depth that it is evident no large tumour is present, although a casual and perfunctory examination may lead to the diagnosis of an ovarian tumour. Bimanual examination, if necessary under an anæsthetic, will usually clinch the diagnosis.

There remain to be considered some less common conditions which may be confused with ovarian tumours.

In cases of *tuberculous peritonitis* where there is a considerable quantity of fluid encysted between adherent coils of gut, a mistake may be made if abdominal examination is not thorough. Intestine adherent to the anterior wall of an ovarian cyst is one of the rarest conditions met with in gynecology, but in most cases where there is encysted serum in tuberculous peritonitis, light percussion will bring out a resonant note over some part of the anterior surface of the swelling. It is common in such cases to find on vaginal examination evidence of tuberculous disease of the tubes.

A large *hæmatocele* or an *encysted collection of pus or serum* may give rise to an abdominal swelling which may be mistaken for an ovarian cyst. The main points in the diagnosis, apart from the history, are that the encysted collection of fluid is tender on palpation, fixed, and, at any rate over part of its surface, resonant on percussion.

A large *renal swelling*, most commonly hydronephrosis, may be mistaken for an ovarian tumour. The history may be of some assistance in making a diagnosis. The chief points are that the kidney tumour cannot be separated from the loin, that a line of colonic resonance can be found running over it, although the colon may be so much flattened as not always to give a resonant note, that no resonance can be obtained behind the tumour, and that on bimanual examination, possibly only under an anæsthetic, the uterus can be felt distinct from the tumour. Sometimes both ovaries may be felt under an anæsthetic. This applies to all the tumours that are still to be mentioned.

A *hydatid cyst of the liver* has often been mistaken for an ovarian cyst, and if it fills the whole of the abdomen the diagnosis must be very difficult. A *large distended gall-bladder*

may also have to be distinguished from an ovarian cyst. As a rule, however, an hepatic tumour does not extend down to the brim of the pelvis and descends with the liver on respiration.

Splenic tumours do not often give much trouble in diagnosis, even though they may descend into the pelvis, since their attachment is usually, and their shape practically always, characteristic.

Omental and mesenteric cysts may resemble ovarian cysts so closely that the correct diagnosis is not made until the abdomen is opened, but in many cases the free range of mobility, absence of connexion with the pelvis, and the results of percussion will suggest the probable nature of the tumour.

Pancreatic cysts are rare. They usually push the stomach upwards and the transverse colon downwards. Percussion will then show resonance below the tumour, and this practically excludes an ovarian origin. If the transverse colon lies above a pancreatic cyst, a mistake in diagnosis is likely to be made.

Treatment.—There is only one rule for dealing with ovarian tumours, viz. to remove them as soon as they are discovered, except in some cases in which the patient is pregnant (see p. 449). The reasons for this rule are:—

1. No tumour can be pronounced with certainty to be innocent until it has been removed and examined.

2. Some ovarian tumours, although innocent, will grow until they kill the patient.

3. Ovarian tumours are always liable to accidents—torsion of the pedicle, suppuration, etc.—which involve danger to life.

Fortunately, we can say with confidence that the risk to the patient is less if she has an ovariectomy performed than if she is not operated on. An uncomplicated ovariectomy is one of the safest of abdominal operations, and the patient can be told that if no unexpected complications are found at the operation the chances of her recovery ought to be about 100 to 1. It must be remembered, however, that the surgeon can never be certain that an ovariectomy will be uncomplicated until he is performing the operation, and that adhesions, deep burrowing of the tumour, etc., may make ovariectomy one of the most difficult of operations. However, the present-day mortality after ovariectomy, even when complicated and difficult, barring advanced malignant disease, is very low. The patient's consent to the operator having a free hand

should always be obtained before the operation. This means that he is to be allowed to remove anything that he considers should be removed. With a large ovarian tumour present it is often, if not usually, impossible to say beforehand that the other ovary is not also cystic. Before the operation, again, it is seldom, if ever, wise to assure a patient with a large ovarian tumour that the other ovary is healthy and that she may bear children after the operation. Nor is it ever wise to assure a patient before the operation that her tumour is perfectly innocent. Especially in the case of young girls and of middle-aged and elderly women it is well to warn some relative before the operation that the tumour may not be innocent. Fortunately, in the majority of cases the surgeon can say immediately after the operation that there is no fear of any recurrence of the trouble unless a tumour forms in the remaining ovary, though in some cases he will have to say that he must wait for microscopic evidence.

Tapping an ovarian cyst without making an abdominal incision is an obsolete procedure.

Treatment of the other ovary.—During the operation for removal of an ovarian tumour the condition of the other ovary should always be inspected, and in an elderly or middle-aged woman it should be removed if it shows distinct signs of cystic change. A younger woman, particularly if only recently married or about to be married, should, if possible, be given a chance of becoming a mother, even if she has to have another ovariectomy performed in a few years' time.

If the patient is young, an attempt should be made to leave her at least part of one ovary. If the whole organ cannot be left, part of it should be left after "resection," i.e. excision of the diseased part, leaving behind a portion of the oöphoron, with bleeding-points ligatured or sutured with scrupulous care, since there is a danger of hæmorrhage occurring after this operation. In a certain proportion of cases in which resection of the ovary is carried out, it is necessary later on to perform a further operation for removal of an ovarian tumour which has grown from the part of the ovary left behind. In cases where resection is impracticable, ovarian grafting may be performed, a portion of the oöphoron being sutured to the raw surface in the broad ligament.

If a tumour is found to be malignant, the other ovary and the uterus should be removed as well. Removal of a healthy ovary in a

young woman because the other ovary was cystic has been recommended, but it is strongly to be deprecated. Whether the other ovary, if healthy, should be left after removal of a papillomatous ovarian tumour, depends upon circumstances. If the patient is middle-aged, removal of the other ovary and of the uterus is to be advised, but this drastic procedure is not to be recommended in the case of a young woman. Several cases have been recorded of patients remaining in good health and bearing children after removal of a papillomatous ovarian tumour. If there is doubt whether the other ovary is healthy or not, Gottschalk advises that it be split for purposes of examination. If it proves to be healthy it is sewn up with catgut and left in its place.

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OVARIAN DERMIDS (see OVARIAN CYSTS).

OVARIAN FIBROMATA.—Most solid tumours of the ovary are malignant. The only exceptions are the fibromata, which are said to represent 2 per cent. of all ovarian tumours. Three varieties are described. In the first, the tumour represents the whole ovary. In the second, the fibroma is a tumour of only part of the ovary, the rest of which may be more or less normal or may be greatly stretched to form a capsule; sometimes the tumour can be shelled out of its capsule of ovarian tissue. In the third variety, of much less importance, there are one or more small, sometimes pedunculated fibromata growing from the surface of the ovary.

The first and second varieties can be considered together. They form tumours which grow slowly but may attain a considerable size, weighing several pounds. The surface is smooth or raised up into rounded elevations. The colour is usually a dead-white, sometimes slightly pink. Unless very degenerate, they are extremely hard, white on section, showing a rather homogeneous or fibrous structure, with sometimes a whorled appearance, although this is usually much less distinct than in uterine fibroids. Their hardness and density are characteristic, and often help in the clinical diagnosis from uterine fibroids. A uterine fibroid may be as hard, but only if it is undergoing calcareous degeneration, in which case it generally gives a characteristic sensation to the examining hand. Ovarian fibromata often degenerate, undergoing oedematous and myxo-

OVARIAN FIBROMATA

matous changes, the tissue becoming softer with formation of small cystic spaces. Unlike ovarian sarcomata, they rarely show hæmorrhagic areas on section. While ascites in sufficient amount to be diagnosed clinically is rarely found with innocent cystic ovarian tumours, except papillomatous cysts, or with uterine fibroids, it is a common occurrence with ovarian fibromata. Sometimes free fluid is poured out rapidly while the patient is under observation.

Microscopically, the tumours are found to be composed of interlacing bundles of cells and fibres in varying proportions, with well-formed blood-vessels. To determine whether a section should be called fibroma or sarcoma is often a task for a skilled microscopist, especially if the tissues stain badly as the result of degeneration or interference with nutrition from acute torsion of the pedicle.

In the third variety, one or more hard tumours, usually small, often not much larger than peas, project on the surface of the ovary, or are attached by pedicles. They are seldom large enough to be of clinical importance.

Diagnosis.—The differential diagnosis in these cases lies between three tumours, one very common *uterine fibroid*, and two much rarer—*ovarian sarcoma* and *fibroma*. *Sarcoma* grows more rapidly than *fibroma* of the ovary, but the history of rate of growth is seldom of much importance, since most patients are bad observers. Cachexia would point to *sarcoma*, ascites to either *sarcoma* or *ovarian fibroma*; if the tumour is exceedingly hard, to *fibroma*; if softer, to *sarcoma*. As was said above, it is rare to find a smooth uterine fibroid, i.e. one not affected by deposition of lime salts (calcareous degeneration), which gives to the hand a suggestion of the hardness and unyielding density characteristic of *ovarian fibromata*. Bimanual examination may not give much help, since a pedunculated subperitoneal uterine fibroid may have as much range of movement apart from the uterus as an *ovarian fibroma* or *sarcoma*. If there are multiple tumours they are probably uterine.

To sum up: a very hard movable unilateral tumour, not intimately connected with the uterus, and associated with ascites but not with cachexia, is probably an *ovarian fibroma*. A solid movable tumour (or solid bilateral tumour) not intimately connected with the uterus, associated with cachexia and loss of flesh, and with or without ascites, is probably an *ovarian sarcoma*.

OVARIAN TUMOURS, MALIGNANT

A solid movable tumour, if not unusually hard, whether intimately connected with the uterus or not, with absence of ascites and cachexia, is probably a uterine fibroid.

In some cases a certain diagnosis between an ovarian tumour and a pedunculated uterine tumour can be made only by operation.

Treatment is by removal.

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OVARIAN PREGNANCY (see PREGNANCY, EXTRA-UTERINE).

OVARIAN TUMOURS, MALIGNANT.

—Under this heading must be considered:

1. CARCINOMA.
2. SARCOMA, including endothelioma, perithelioma, and melanotic sarcoma, which may be classed with the sarcomata for convenience.
3. SOLID TERATOMATA.

1. **Carcinoma of the ovary.** (1) *Primary carcinoma.*—These tumours are rare, but probably they are more common than secondary carcinomatous tumours. They are, as a rule, smooth with a rather irregular outline, and seldom very large, rarely reaching the size of an adult head. When small they are surrounded by a fibrous capsule derived from the tunica albuginea, but during the process of growth this may be invaded until the carcinoma reaches right up to the surface. They are often bilateral, but the tumour on one side may be much larger, and apparently of older growth, than that on the other. They are commonly accompanied by ascites, and in the late stages the peritoneal cavity may be studded with secondary nodules. Secondary growths may also occur in other parts of the body. On section these tumours are soft, more or less homogeneous except that they often contain small cavities, sometimes due to degeneration, in other cases lined by epithelium, and are often rather brain-like in appearance. Microscopically, varying characters may be shown by different parts of the same tumour, some sections exhibiting alveolar structure, while others reveal masses of epithelial cells with very little arrangement. Not uncommonly parts of these tumours are too degenerate to stain well.

(2) *Carcinoma occurring in ovarian cysts* is the most common variety of carcinoma of the ovary. It is most often seen in papillary cysts, fairly frequently in pseudo-mucinous cysts, and less commonly in cystic teratomata. Many papil-

OVARIAN TUMOURS, MALIGNANT

omatous ovarian cysts are carcinomatous. This should be suspected when the papillomatous masses are found to be soft, friable, and vascular, and less fibrous than usual. On section the papillæ are seen to be compound and branching, not simple, and there is a high degree of proliferation of the epithelium of the tufts, sometimes with formation of alveoli. On naked-eye examination of a pseudo-mucinous cyst, the presence of soft, solid tissue, invading the cystic adenomatous tissue, suggests the presence of carcinoma, which may be determined by a microscopic examination.

Carcinoma occurring in teratomata is described at p. 451.

(3) *Secondary carcinoma.*—It has been taught that this is the most common variety of carcinoma of the ovary, the primary growth being usually in the stomach, some part of the intestine, the breast, or the uterus. This is probably true of *carcinoma of the ovary*, but is not true of *carcinomatous tumours of the ovary*. Post-mortem examination of women who have died from carcinoma of the stomach, intestine, breast, or uterus often shows that the ovaries are affected with carcinoma together with most of the contents of the abdominal cavity, the peritoneum being studded almost all over with carcinomatous nodules, but it is comparatively rare to find that there is sufficient enlargement of the ovary for it to be recognized clinically as a tumour. The secondary growth in the ovary may be very large, and is frequently bilateral, while the primary growth may be small and attract no attention clinically. It may be impossible by microscopical examination to decide whether the growth of the ovary is primary or secondary.

2. *Sarcoma of the ovary.*—Sarcomata are found in about 5 per cent. of all ovarian tumours. They are commoner during youth and in late middle life than during the reproductive period, are often bilateral, and may grow to a very large size. They are usually accompanied by ascites. Microscopically, two varieties can be recognized, spindle-celled and round-celled sarcomata.

The *spindle-celled sarcoma*, which are also called *fibro-sarcomata* of the ovary, resemble in appearance the fibromata. They are sometimes very hard. The consistence may vary in different parts of the tumour, some parts being hard and others soft. They are less malignant than the round-celled variety. Microscopically they are composed almost entirely of spindle-cells.

The *round-celled sarcomata* are softer, much more malignant, and are found with comparative frequency in children and young girls. They are more vascular than the spindle-celled sarcomata, either pink or red in colour, are prone to degenerate, and frequently show hemorrhage into the substance of the tumour.

Sarcoma is sometimes found in teratomatous tumours and in the walls of adenomatous cysts.

Endotheliomata, tumours growing from the endothelium of the small vessels, and *peritheliomata*, growing from the tunica adventitia of the small blood-vessels and lymphatic vessels, are comparatively rare ovarian tumours which may be classed between the sarcomata and the carcinomata. They are nodular in outline, soft and spongy on section. They are very malignant. The exact diagnosis can be made only by a skilled microscopist.

Melanoma, or *melanotic sarcoma* or *melano-carcinoma*, occurs infrequently in the ovary, usually secondary, but in very rare cases apparently primary. It has a characteristic appearance, very dark brown or black, suggestive at first sight of congestion from torsion of the pedicle. Microscopically, the alveolar structures and the pigment granules are equally characteristic.

3. *Solid teratomata of the ovary.*—Solid teratomata, some of the least common of ovarian tumours, are best described among malignant ovarian tumours, as they are seldom innocent. It is impossible to draw a hard-and-fast line between the ordinary, common ovarian "dermoid" or cystic teratoma and the much rarer solid teratoma, for the common tumour may contain a good deal of solid tissue while the malignant solid teratoma may possess cystic spaces. They cannot all be said definitely to be malignant, but many of them are, and recurrence may follow removal of a tumour in which no malignant change can be found under the microscope. While the innocent cystic teratoma is of slow growth and seldom attains a size much larger than a fetal head, the more solid tumours may grow rapidly and to a much greater size, and cause ascites and metastatic growths, chiefly in the peritoneal cavity. These metastatic growths may be composed of any of the varieties of cell found in the teratoma, or may be definitely carcinomatous or sarcomatous. A few cases have been described in which ovarian teratomata contained a large amount of tissue closely resembling that of the thyroid gland.

Solid teratomata are found most commonly

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in the second decade, but have occurred sometimes in adult life. Though apparently solid, they are found on section to have cystic spaces in their interior, lined by epithelium. All the three layers—epiblast, mesoblast, and hypoblast—are represented in their structure, but whereas in the innocent cystic teratoma are found well-formed structures such as teeth, bone, skin, with hair-follicles and sweat-glands, cartilage, brain-like matter and even portions of intestine, etc., in the solid teratoma there are masses of embryonic tissue rather than organized structures. This explains their potential malignancy.

Symptomatology and diagnosis of malignant ovarian tumours.—In many cases the diagnosis of malignancy is made only by microscopic examination, and occasionally the proof that part of the tumour is malignant may come as a surprise, as there had been nothing in the clinical features or in the naked-eye examination to suggest malignancy.

Clinically, the malignant character of a tumour is suspected if cachexia and loss of weight have occurred with a tumour whose mere size is not enough to account for these symptoms. Bilateral tumours are more likely to be malignant than innocent. Rapid growth of the tumour, pain and fixation without evidence of infection, and unilateral oedema of the thigh and leg or of the vulva will all suggest malignancy. The presence of several masses in the abdomen, together with ascites, evidence of infiltration of the abdominal wall, and the presence of nodules in the peritoneum of Douglas's pouch, afford strong evidence that the tumour is malignant. Rectal examination may be extremely useful in leading to the detection of infiltration of tissues surrounding the tumour.

Ascites is so rarely found, in appreciable quantity, with innocent ovarian tumours, except fibromata and papillomatous cysts, that its presence must excite suspicion, unless the tumour is freely movable and very hard.

The differential diagnosis between small bilateral malignant ovarian tumours and bilateral *salpingo-oöphoritis* may sometimes be difficult when a good history cannot be obtained. Cachexia and loss of flesh with little rise of temperature will point to the diagnosis of malignant disease. Occasionally the difficulty can only be solved by an exploratory operation.

Treatment.—A diagnosis of malignancy does not necessarily preclude treatment by ovariectomy. If the patient's general condition is

good enough to withstand an operation it may be well worth doing, and she may be free from symptoms pointing to recurrence for months or years. Debility of the patient, fixation of the tumour, palpable secondary growths in the omentum, in the parietal peritoneum or in the peritoneum of Douglas's pouch, contraindicate operation. As a working rule, if there seems to be a reasonable chance of removing the tumour the abdomen should be opened. It is often best, especially in cases in which there is a large amount of ascites, to make a very small incision admitting one finger. If secondary nodules are felt the abdomen is at once closed after the free fluid has escaped, and very little if any harm has been done. If the exploring finger finds no evidence of extension of the growth the incision is enlarged and the abdomen explored by the hand. A thorough exploration should be made in any doubtful case before the operation of removal is begun, for incomplete operations, in which the tumour is torn and part of it left behind, are likely to prove fatal, occasionally by causing bleeding, and often by bringing about what seems to be a toxæmia. Evacuation of a large quantity of free fluid by an exploratory incision may give great relief. Sometimes the fluid accumulates very rapidly again, and repeated tapping may be necessary, but in some cases the abdomen fills up again only slowly.

Occasionally an exploratory incision in a case complicated by ascites may have the happiest results, an innocent tumour, either a fibroma or a papillomatous cyst, which had been thought to be fixed proving to be freely movable and easy of removal, with restoration of the patient to her normal state of health.

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OVARIES, DISPLACEMENTS OF (*see PELVIC ORGANS, FEMALE, DISPLACEMENTS OF*).

OVARIES, TUBERCULOSIS OF (*see SALPINGO-OÖPHORITIS*).

OVERLYING (*see Suffocation, under ASPHYXIA*).

OXALIC ACID, POISONING BY (*see POISONS AND POISONING*).

OXALURIA (*see URINE, EXAMINATION OF*).

OXYCEPHALY (*see SKULL, CONGENITAL ANOMALIES IN SHAPE OF*).

OZÆNA (*see RHINITIS*).

PACHYDERMIA LARYNGIS (*see* LARYNGITIS, CHRONIC).

PACHYMENINGITIS (*see* MENINGITIS).

PAGET'S DISEASE OF BONE (*see* OSTEOITIS DEFORMANS).

PAGET'S DISEASE OF THE NIPPLE (*see* BREAST, AFFECTIONS OF).

PAIN, ABDOMINAL (*see* ABDOMINAL PAIN, DIAGNOSIS OF).

PAIN, CENTRAL.—By central pain is meant pain produced directly by lesions or diseases of the central nervous system, but referred peripherally or to the surface of the body. It is rare, for central nervous diseases run, as a rule, a painless course unless the extramedullary portions of the afferent roots are also involved; foci of inflammation, as encephalitis or myelitis, tumours, and even abscesses of the brain and cord, usually produce pain only when they lead to an increase of intracranial pressure or involve sensory fibres of the peripheral system.

The central conditions that generally cause peripherally-projected pain may be divided into two classes—(1) those that do so by irritating the pain-conducting tracts, and (2) disease of the optic thalamus, in which, as we shall see, the cause of the pain is different.

1. Irritation of the pain-conducting tracts.—Pain is occasionally a result of partial injuries of the pain-conducting fibres of the *ventro-lateral columns of the cord*; in a considerable proportion of the cases that have been observed it followed a local traumatic lesion. A man recently under my care is a good example. Two and a half years ago he received a slight injury to his back, and a few months later began to suffer with severe burning and tearing pains in his right leg and on this side of his body up to the nipple. The pains were variable—sometimes very intense, at other times only slight. He had a characteristic Brown-Séquard paralysis, his left leg being weak and spastic, and pain and thermal sensibility being reduced on the right side below the level of the nipple. During the late War I also observed a large number of cases in which, as a result of gunshot wounds of the spine, severe spontaneous pains occurred below the level of the lesion, but these pains were only

temporary, persisting at the most for four or five weeks. They were generally unilateral, and then, as in the case just mentioned, sensibility to pain was lost or diminished in those areas to which the patient referred them.

Similar pains are not uncommon in *syringomyelia*; they may be limited to a zone round the trunk or may spread over a whole limb. As a rule, they occur in regions in which analgesia is developing, and may therefore be explained by the progressive affection of the pain-conducting fibres of the cord.

But such pains due to irritant lesions are most commonly met with in diseases of the *sensory tracts of the brain-stem*. In a case recorded by Mann, for example, a softening in the right half of the medulla was the cause of severe burning and shooting pains throughout the left limbs, the left side of the trunk, and in the right face. A similar condition may be produced by disease of the pons, or midbrain. A man at present under my care with symptoms of a lesion of the left fillet in the mid-brain complains of sharp burning pains in the right side of his body, sometimes in the leg, sometimes in the arm, but more frequently in the right half of his trunk.

Pain of this origin has certain distinguishing features; though more or less constant, it varies considerably in severity, it is independent of peripheral excitations, and is little if at all influenced by drugs. We are, in fact, more or less helpless in its treatment, though when it is due to a spinal lesion relief may be obtained by section of the ventro-lateral column of the cord above the level of the lesion—an operation which is, however, attended by considerable risk.

2. The thalamic syndrome.—Dejerine and Roussey originally described under this title a group of symptoms due to lesions of the lateral portion of the optic thalamus, and of these pain in the opposite side of the body is the most prominent. Lesions of vascular origin are its most common cause; the usual history is of a stroke that produced a slight or transient hemiplegia and, as a rule, a large amount of sensory disturbance. Within a few weeks the patient begins to suffer with persistent or paroxysmal pains, often of extreme severity, which are generally referred to one half of the body, but they may be limited to any part of the affected side. They are

PALATE, PARALYSIS OF

excited and aggravated by various external influences, particularly by such as normally produce discomfort, and in fact, though apparently spontaneous, they are largely dependent on peripheral stimuli. The prick of a pin, a pinch, a scrape, contact with a cold object, or even a draught of cold air may excite intense pain. On the other hand, the avoidance of such stimuli may allow the patient to remain fairly comfortable. As this condition is often associated with a pleasurable over-reaction to stimuli that naturally excite sensations endowed with pleasurable-feeling tone, an over-reaction to all affective stimuli may be regarded as a characteristic feature of it. In this respect it differs from those conditions in which pain is due to lesions of the cord or the brain-stem. The pains of thalamic disease are not, therefore, as has been generally assumed, due to irritation of the central conducting tracts, but are a result of the removal of the inhibitory control which the cerebral cortex normally exerts on subcortical centres that are concerned in the perception of this form of sensation.

These positive sensory symptoms are consequently comparable to the motor hyper-tonicity produced by cerebral lesions, which is now recognized to be a result of the removal of the inhibition that the brain normally exerts on subcortical motor centres.

In addition to the sensory disturbances, and the pains and the slight motor paresis that usually accompany them, there are frequently involuntary movements of the tremulous or choreic type of the affected limbs.

Little, unfortunately, can be done to relieve the pain; drugs are of little use, except morphia, and this had better be avoided, but precautions against exposure to cold and other peripheral stimuli may enable the patient to live in comparative comfort.

GORDON HOLMES.

PAIN, REFERRED (see NEURALGIA).

PAINTER'S COLIC (see Chronic Lead Poisoning, under POISONS AND POISONING, p. 600).

PALATE, PARALYSIS OF.—The muscles of the palate are innervated mainly by those fibres of the spinal-accessory nerve that join the vagus. A unilateral palsy is indicated by drooping of the same side of the palatal arch during rest, while in movement, as in phonating "Ah," the raphe is drawn towards the unaffected side. The palatal

PANCREAS, CARCINOMA OF

reflex is also lost on the paralysed side. The unilateral form has little clinical importance, but bilateral paralysis interferes with swallowing, gives articulation an unmistakable nasal character, and allows food to enter and regurgitate through the nose.

Palsy of the palate may be due to a neuritis of its motor nerves; this is especially common in diphtheria, and it occurs occasionally with other septic infections of the mouth and nose. In bulbar paralysis the affection of the palate plays a large part in the difficulty in articulation and swallowing. One or both sides are not uncommonly affected, together with the tongue and larynx, by syphilitic meningitis and other lesions of the medulla oblongata.

GORDON HOLMES.

PALPITATION (see HEART, PALPITATION OF).

PALSY, FACIAL (see FACIAL PALSY).

PANCREAS, CARCINOMA OF.—The pancreas, like any other organ, may be the seat of new growth, but the large majority of cases of carcinoma are of secondary origin. The primary focus occurring in an adjacent organ, for example carcinoma of the pylorus or of the posterior wall of the stomach, frequently involves the head of the pancreas.

Etiology.—Carcinoma of the pancreas is a comparatively rare disease. In Bashford's statistics one per cent. of all cases of carcinoma were primary new growths in the pancreas. The disease is about twice as common in man as in woman, and the age-incidence is that of carcinoma elsewhere.

Pathology.—The growth is most frequently situated in the head of the gland, rarely in the tail. Usually the tumour is hard, consisting of a fibrous matrix with alveoli of cancer-cells. More rarely it is soft or colloid. Attempts have been made to determine the seat of origin (whether ducts, alveoli, or islands of Langerhans) according to the type of new growth, but so far no definite relationship has been traced.

Symptomatology.—The disease begins as a rule with indefinite symptoms, such as epigastric pain and general digestive disturbances. Pain is a constant symptom, and may be continuous or intermittent. In the latter event it is thought to be due to pressure on the liver causing obstruction to the outflow of secretions. Jaundice almost always develops, due to pressure on the bile-duct. It may appear suddenly, simulating cholelithiasis, but

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the jaundice shows no intermission, and increases in severity to the stage of "black jaundice." The gall-bladder is usually distended, and the liver may be enlarged. Digestive disturbances result in fatty stools and in the presence of muscle-fibres in the fæces, changes which can be referred to the insufficiency of pancreatic ferments, and there are wasting and emaciation, which become progressively more rapid as the disease develops. A tumour in the epigastrium may be found, but is not a constant sign. Other symptoms, such as ascites and œdema of the legs, appear later, and can be traced to the developing mass.

Diagnosis.—In a middle-aged or older subject progressive jaundice associated with a distended gall-bladder and with the presence of a tumour in the epigastrium points to carcinoma of the pancreas. In *gall-stones* the jaundice is intermittent and the gall-bladder usually contracted. Rapid cachexia is a valuable sign, while the temperature, which is frequently raised in cholelithiasis, is generally subnormal in carcinoma of the pancreas.

Prognosis.—In the pancreas, carcinoma tends to be more rapidly fatal than in other organs. Death usually occurs from four to six months after the onset of severe symptoms.

Treatment.—Removal of tumours of the pancreas has been carried out, but, although, the patient may survive the operation, death usually occurs within a few months. The treatment resolves itself into measures for the relief of symptoms. Cholecystenterostomy for the relief of jaundice has been performed, but as a rule the weakness of the patient precludes major surgery. Administration of pancreatin or of an emulsion prepared from the fresh pancreas of the pig has been recommended, and a diabetic diet, which makes the least call on the activities of the gland, will probably give most relief to the patient.

C. M. WILSON.

PANCREAS, CYSTS OF.—The term cyst of the pancreas includes various lesions which have this feature in common, that in the upper part of the abdominal cavity there is a collection of fluid surrounded by a capsule which is derived wholly or in part from the tissues of the pancreas.

Etiology.—Statements as to the sex-incidence of pancreatic cysts are conflicting; on the whole, they appear to be more common in women than in men. The large majority of recorded cases have occurred in early middle life. A cyst, however, has been noted in a

child aged 13 months, and in a few cases in patients over 60 years.

Pathology.—Cysts of the pancreas may be formed when for any reason there is an obstruction to the overflow of pancreatic secretions. Thus they may be found in conjunction with pancreatic calculi, or with chronic interstitial pancreatitis, where there is occlusion of branches of the pancreatic duct by the formation of fibrous tissue. Artificial ligation of the duct is followed by very little dilatation of the duct behind the ligature, and gives rise to inflammatory changes only, and total occlusion in man is usually followed by similar changes. But it is thought that partial or intermittent occlusion may favour the formation of cysts.

Proliferation cysts are of rare occurrence. A few cases, mainly in women, have been described in which multilocular cysts resembling cystic adenomata of the ovary have been found in connexion with the pancreas; they are thought to arise by proliferation of the epithelial cells followed by accumulation of fluid in the cavities thus formed.

Cysts may follow injury, and have been noted after cases of acute hæmorrhagic pancreatitis. The pathogenesis of the lesions in these cases is probably similar. The injured tissue undergoes solution, and is subsequently encapsulated by fibrous tissue.

Cases also occur in which collections of fluid are surrounded by connective tissue, but without an epithelial lining. Such pseudo-cysts are of more frequent occurrence than true cysts.

The contents of the cyst give little or no evidence of the cause of the lesion. The fluid may be clear and watery, but is usually mixed with fresh or altered blood, even when there is no history of injury to the gland. As a rule, ferments are present, but the presence or absence of these does not possess the diagnostic value formerly assigned to it, since the secretion of an inflamed pancreas may contain little or no ferment, while conversely fat-splitting and diastatic ferments may occur in fluids which do not arise from the pancreas.

Symptomatology.—The physical signs are by no means constant, since pancreatic cysts can arise in different parts of the gland, and according to their position will bear different relationships to surrounding organs. As a rule, a spherical tumour which fluctuates on palpation, situated in the epigastrium, but lying behind the stomach, suggests a pancreatic cyst. It must be remembered that small cysts may be impalpable, being com-

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pletely covered by the stomach, while large cysts may occupy the lower part of the abdominal cavity and be confused with cysts of the pelvic organs. Instances have been noted in which a palpable tumour has suddenly disappeared, while at the same time there was a sudden onset of diarrhoea, pointing to rupture of the cyst and discharge of the contents into the intestine. Other cysts have been known to rupture into the peritoneal cavity.

The **symptoms** are variable. As a rule there are digestive disturbances, with loss of flesh and deep-seated epigastric pain. There may be pale, bulky stools, and undigested muscle-fibres may be detected in the faeces, indicating a lack of the pancreatic ferments. Jaundice is rare, since a cyst seldom occupies the head of the gland. Other symptoms are due to the pressure of the growing cyst upon adjacent organs. Thus pressure on the inferior vena cava may cause oedema of the lower extremities, and ascites follows pressure on the portal vein. Pressure on the stomach results in discomfort after eating, with vomiting, and pressure on the intestines gives rise to colicky pain and constipation.

Diagnosis.—The presence of a fluctuating tumour is the most important sign in diagnosis. In determining its origin, its relations to the stomach and colon are of the greatest significance. It lies behind these, and in difficult cases can be defined by inflation of the stomach, followed by palpation. In such a case the dullness of the cyst will probably pass behind the stomach, which obliterates it, thus distinguishing it from a *distended gall-bladder* and from a *cyst of the liver*. Albu finds that X-ray examination gives a characteristic picture, the stomach making a half-circle round the tumour, beginning at its top left-hand side.

Prognosis.—Usually, if there is no surgical interference, the disease causing the cyst steadily progresses. If the cyst is not drained, death may result from rupture of the cyst, causing general peritonitis, or there may be progressive atrophy of the gland, causing diabetes, or death may ensue from pressure symptoms. Operation for drainage of a cyst is successful in the great majority of cases, and recurrence is rare.

Treatment.—Tapping a pancreatic cyst is to be strongly deprecated, since there is great danger of puncturing other organs and of setting free an irritant fluid in the peritoneal cavity. The treatment should consist in free

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incision and drainage. In cases in which there are only slight adhesions to neighbouring organs, removal is possible and has been attended with success, but these cases are, unfortunately, rare. Drainage is very apt to be followed by fistulae, which may persist for months, and even in one recorded case for three years. After operation, convalescence is shortened by placing the patient on a diet such as is given in diabetes, which reduces the calls upon the pancreas to a minimum.

C. M. WILSON.

PANCREATIC CALCULI.—The presence of concretions in the ducts of the pancreas is of rare occurrence, only two cases being noted in a series of 1,500 post-mortem examinations at the Johns Hopkins Hospital. In contrast to gall-stones, they occur more frequently in men than in women.

Pathology.—Pancreatic calculi are found on analysis to consist mainly of inorganic salts, in particular of calcium phosphate and calcium carbonate; they occasionally contain magnesium in small quantities. They are situated most frequently in the larger ducts, the usual site being the duct of Wirsung. They are never found in a healthy pancreas, and are accompanied by bacterial infection. It is not yet established whether the infection is the cause of calculus or is secondary to it. Blocking of the duct gives rise to an accumulation of the pancreatic secretion, and dilatation of the duct behind the obstruction is often noted. Inflammatory changes take place, and are in the course of time replaced by scar tissue, or more rarely a cyst may be formed. In cases of acute infection a pancreatic abscess sometimes develops.

Symptomatology.—Small pancreatic calculi may give rise to neither signs nor symptoms and may be discovered only at necropsy. Larger calculi cause symptoms closely resembling those produced by gall-stone. There is epigastric pain, which may be persistent or may resemble colic, and is associated with the passage of the calculus along the duct. The calculus may subsequently be passed per rectum. The pain, like that of gall-stone colic, is often accompanied by vomiting and collapse. There may be fever due to bacterial invasion of the common duct. If a calculus finds its way to the diverticulum of Vater it occludes the opening of the bile-duct and may give rise to temporary jaundice. If the disease is of long standing and the blocking of the ducts has

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produced chronic inflammation, there is impairment of the functions of the pancreas, which is reflected in the presence of undigested fat and muscle-fibres in the stools, while later glycosuria develops, due to the invasion of the islands of Langerhans and the failure of the internal secretion.

Diagnosis.—The difficulty lies in distinguishing a case of pancreatic calculus from one of *cholelithiasis*, since the signs and symptoms are so much alike. Robson and Cammidge claim that an X-ray examination is of great value, since gall-stones are not opaque to the rays while the pancreatic calculi are clearly shown. The presence of concretions in the faeces, which on analysis are found to consist for the most part of calcium carbonate, is a valuable sign, while the later effects of impaired fat-digestion and diabetes indicate a lesion of the gland.

Prognosis.—Pancreatic calculi may not set up any signs or symptoms, and may only be detected after death from some other disease. On the other hand, they may give rise to inflammation, which in time may involve the greater part of the gland, and death may result from impaired metabolism and diabetes.

Treatment.—The literature of pancreatic calculus is so small that it is difficult to compare different therapeutic methods. Medical treatment is for the most part directed to the relief of the pain by sedatives and to measures such as the giving of quantities of warm water acidified with carbon dioxide in order to promote the activity of the gland, with a view to dislodging the obstruction. Surgical treatment for the removal of the calculus has been undertaken, and in some cases has been followed by recovery without recurrence of the symptoms.

C. M. WILSON.

PANCREATIC DIABETES (*see* DIABETES MELLITUS).

PANCREATITIS, ACUTE.—It has been customary to classify the cases of acute disease of the pancreas under three headings—(1) Acute Hæmorrhagic, (2) Gangrenous, (3) Suppurative Pancreatitis; and in the early history of the disease these were distinguished as clinical entities. The groups are in reality only three stages of one and the same condition, but the classification, which was originally suggested by Fitz, serves a useful purpose in distinguishing the phases of an acute lesion of the gland.

Etiology.—Acute hæmorrhagic pancreatitis may develop as the result of direct injury to the gland, but is usually of spontaneous origin and occurs in subjects of all ages. It is more frequently met with in men than in women, and generally its victims are persons who have previously been in good health. The obese are said to be most liable to the disease, and heavy eating and drinking with disorders of the liver and gall-bladder are claimed as predisposing factors.

Pathology.—The pathology of acute pancreatitis is of considerable interest and much has been written on the subject in the last few years. The disease may be primary or secondary to an infection which has reached the gland from some distant focus. It is found in association with lesions so diverse as diseases of the gall-bladder and ducts, duodenal ulcer, colitis, appendicitis, and inflammatory affections of the pelvic organs. Formerly it was thought to be due in all cases to infection from the biliary tract, which is rendered possible by the anatomical conditions in the ampulla of Vater. Recent investigation seems to point to the conclusion that a more distant infection may be responsible, spreading by way of the lymphatics which anastomose in the retro-peritoneal tissue of the pancreas. But it seems clear that the majority of cases are associated in some way with the passage of bile into the pancreas. In one case of fatal pancreatitis described by Opie, this was undoubtedly brought about by occlusion of the common duct by a gall-stone which was of just such a size that, while blocking the common orifice, it allowed free passage between the bile-duct and the duct of Wirsung. This discovery led to the belief that the majority of cases of pancreatitis were associated with the presence of gall-stones, a conclusion that has not been borne out by the facts available. Experimentally, it has been shown that hæmorrhage into the pancreas can be caused by injection of bile or of intestinal contents or of dilute hydrochloric acid into the ducts. The explanation of the action of the acid is probably that it causes temporary spasm of the common duct sphincter, so that there is an increase of pressure in the bile-duct, leading to a flow of bile into the pancreas. Archibald believes that this spasm of the sphincter is the cause of the majority of cases of pancreatitis. Others, notably Laplace, hold that the condition is infective. *B. coli* and streptococcus among other bacteria have been found in the gland, but many think that

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the presence of such bacteria is secondary to chemical action.

It suffices that, through some irritant agent, trypsinogen is probably liberated in the gland. Mellanby and Woolley have established the fact that *B. coli* has the power of activating trypsinogen, and Vernon has shown that another ferment is present in zymogen form in the pancreatic ferment, which is activated by free trypsin, and which is then much more active than enterokinase: the trypsin set free acts upon the cells of the gland tissue, disintegrating the substance of the gland and setting free pancreatic ferments into the peritoneal cavity, causing profuse hæmorrhage. If operation is performed at this stage the pancreas is found to be enlarged and friable, and frequently presents a mottled appearance due to the juxtaposition of the hæmorrhagic necrotic substance of the gland and relatively normal areas. The affected part is completely broken down, the cells being transformed into a homogenous structure without nuclei. There is usually blood in the peritoneal cavity. At the same time, opaque yellowish-white foci of fat necrosis are found in the fatty tissue, being especially abundant in the neighbourhood of the gland, but spreading to quite distant parts. The disintegration of the pancreatic tissue sets free lipase into the peritoneal cavity, which acts on the stored fat, splitting it into glycerin and fatty acids. The glycerin is absorbed, while the action of the calcium salts of the blood on the fatty acids gives rise to the yellowish-white patches of fat necrosis. The symptoms of collapse may be due to the pressure of the enlarged gland upon the celiac plexus.

Should the patient survive the acute hæmorrhagic stage, a second group of symptoms supervenes; these are due to a peculiar type of toxæmia, the origin of which is obscure. It has been shown by Whipple and others that in cases of acute intestinal obstruction a substance of extreme toxicity is produced which has been identified as a primary proteose. Since the symptoms associated with pancreatitis so closely resemble those of intestinal obstruction, it seems probable that the same substance may be produced in this lesion. Others trace the toxic symptoms to some substance unrecognized which is liberated by the action of the pancreatic enzymes upon the substance of the gland.

If the patient recover from the attack of acute hæmorrhagic pancreatitis, about the fourth day the gland becomes gangrenous, and

generally suppuration supervenes. Purulent fluid may be disseminated throughout the gland, or a circumscribed abscess, which may or may not be surrounded with a fibrous capsule, is formed. Such an abscess may rupture into a peritoneal cavity, causing death from peritonitis.

Symptomatology.—The onset of acute pancreatitis is dramatically sudden. An otherwise healthy person who has perhaps been subject at intervals to slight digestive disorders is suddenly seized with acute pain in the epigastrium, localized just above the umbilicus. Pain in the back radiating to the legs is not uncommon. The epigastric pain is intense, and is associated with vomiting, the vomit as a rule being copious and bilestained and in some instances dark brown or mixed with blood. The patient is collapsed and shows signs of shock. Constipation is present, accompanied by abdominal distension and intense tenderness. At a later stage the patient may become cyanosed and livid, with hurried and shallow respirations and a thready and almost impalpable pulse. In the stage of collapse the temperature is subnormal. Death may ensue within a few hours or days, but if the patient survives the first few days a stage of gangrenous pancreatitis supervenes. The more acute symptoms subside, though pain and tenderness localized in the midepigastrium are still present. At the same time an abdominal tumour becomes palpable. The percussion note over this tumour is tympanitic, due to the fact that the stomach and colon overlie it. With the onset of gangrene the temperature rises. The bowels are no longer confined, and there may be diarrhœa. The third stage, suppurative pancreatitis, begins about two weeks after the onset of the acute symptoms. Suppurative pancreatitis has likewise been described following direct infection when there has been no hæmorrhage into the gland. A localized abscess may be formed, or there may be many disseminated centres of suppuration; in a few cases the whole tissue of the pancreas is found infiltrated with purulent fluid. There is diarrhœa or constipation, more frequently the former, with fever and occasional chills. An abdominal tumour has been reported in a fair number of cases, situated in the midepigastrium behind the stomach and colon. Symptoms referable to the stomach and liver are often present, jaundice being not infrequent.

The operative findings of acute pancreatitis are characteristic, the salient feature being the

presence of numerous fat-necroses in the mesentery and omentum, and even in the pericardium. This condition is pathognomonic of pancreatitis, and is a valuable aid in diagnosis. Associated with the fat-necroses a peculiar "beef-broth" fluid is present in the peritoneal cavity, and the character of the omentum is changed, its condition being described as "boggy." The small intestine is often cyanosed, and the pancreas enlarged. In the hæmorrhagic stage the gland is mottled red and black, and is friable.

Diagnosis. The recognition of acute pancreatitis is dependent on a group of symptoms which may be arranged in two categories according to the time of their appearance. The earlier are secondary to the pressure of the swollen gland and its hæmorrhagic exudate, the later are due to a condition of acute toxæmia that is met with in certain other disorders, and is discussed under Pathology.

The pain in acute pancreatitis is more severe than that met with in cases of perforated gastric or duodenal ulcer, and is situated deep in the epigastrium, radiating to the back. It is never referred to the right shoulder—a useful point in distinguishing it from the pain due to *cholelithiasis*. The patient lies still in bed, in contrast with the victim of *renal colic*. Tenderness and rigidity of the abdomen are not present to the degree common in cases of a perforated viscus. The presence, not less than three days after the onset of the acute stage, of an abdominal tumour, with tenderness in the left costo-vertebral angle, is a valuable diagnostic sign. The normal or subnormal temperature, only becoming elevated after some days, helps to differentiate the disease from a condition of *spreading peritonitis*. The conditions most difficult to separate off from acute pancreatitis are *acute intestinal obstruction* and *gall-stones*, but if laparotomy be accepted as the proper procedure in all three conditions, the importance of making a correct diagnosis early in the disease is less vital.

Prognosis.—If the early toxæmia is not fatal, a general improvement follows, the more acute symptoms subside, and a part of the gland may escape infection or may recover. The most affected part becomes necrotic and finally suppurates, and as a rule sepsis intervenes and, unless relieved, causes the death of the patient. The course of the disease therefore depends on the early recognition and prompt operative treatment of the mild and moderately severe cases, since free drainage may

prevent an unfavourable issue. The mortality following operation has fallen sharply in the last few years. Linder, for example, quotes a series of 16 cases operated on up to June, 1915, where the mortality was 42 per cent., while in a subsequent series of 15 cases, where a diagnosis was made in 75 per cent. of the cases, the mortality was only 13 per cent.

Treatment.—The treatment of all cases is surgical, and the sooner the operation is performed after the onset of the symptoms the greater is the patient's chance of recovery. When suppuration has occurred, the possibility of successful drainage is very remote, since the abscess is seldom circumscribed, and the only cases in which early operation is inadvisable are :

(1) Those in which the collapse and shock are so profound that the patient is obviously unfit for operation. In these cases, measures to combat the shock must be taken with a view to operation later.

(2) Those in which the symptoms are obviously subsiding, and the disease is subacute; here the operation can be postponed until the patient is in a more satisfactory condition.

The pancreas is exposed by an anterior incision, either through the gastro-colic omentum or through the gastro-hepatic ligament, and is freely incised in a longitudinal direction, or numerous blunt punctures are made. Free drainage is established and a tube left in for a considerable time. After recovery from the operation, the patient should be kept on a strictly anti-diabetic diet, and sodium carbonate should be given, so that the secretory power of the gland is taxed as little as possible. The patient should be kept under observation for several years, as a recurrence of symptoms has been noted in some cases, and chronic interstitial pancreatitis may supervene.

C. M. WILSON.

PANCREATITIS, CHRONIC INTERSTITIAL. Etiology.—Chronic inflammation of the pancreas is a disease which is limited to middle life. Its incidence is greatest between the ages of 50 and 60, but cases have been noted occasionally in children and in those who have reached old age. It appears to be equally common in men and in women.

Pathology.—The precise cause of chronic pancreatitis is much in dispute. It is due to an infection, and various bacteria have been isolated from the diseased gland. But the

PANCREATITIS, CHRONIC INTERSTITIAL

means whereby the infection reaches the pancreas and its source of origin are questions that still await a definite answer. In the majority of cases pancreatitis is associated with disease of the gall-bladder and bile-passages, and it has been commonly assumed that cholelithiasis is the main factor. This assumption has been criticized by a number of observers, some of whom maintain that in most cases of chronic pancreatitis there is no associated cholelithiasis. If this be so, we are left with only one definite fact upon which to base theories of causation—the often-quoted case of Opie, who found at operation in a case of chronic pancreatitis that a gall-stone had become lodged in the ampulla of Vater in such a position as to occlude the orifice leading to the intestine but leaving a free passage from the gall-duct to the pancreatic duct. He therefore suggested that the inflammation of the pancreas was due to the passage of bile into the gland. Flexner found experimentally that, while injection of the bile-salts alone into the pancreas had a violently irritant action, the mucin of the bile had a protective action, and he put forward these results as an explanation of the fact that where bile is passed back into the intestine the resulting condition is one of chronic and not of acute hæmorrhagic pancreatitis. More recently, Archibald has suggested that since cases such as that of Opie must be of very rare occurrence, the condition only being possible when the stone is of a certain exact size, the explanation is to be found in a periodical spasm of the sphincter of the common duct at the entrance to the intestine. Such spasm, which he produced experimentally, would lead to an increase of pressure in the bile-passages and to a back-flow of bile into the pancreas. There is, however, another channel of infection which is often neglected in the discussions upon this subject. Many years ago Sappey demonstrated the existence of lymphatic channels linking up the gall-bladder and the pancreas, in the head of which there are numerous small patches of lymphatic tissue. It may be that infection from the gall-bladder tracks along these channels and that the inflammatory changes begin in the lymph-nodes in the head of the pancreas. Whatever the original source of infection and the channel by means of which it spreads to the organ, we find an inflammation of the gland which results in functional impairment of a severity proportional to the amount of the glandular tissue involved.

Symptomatology.—The clinical picture of chronic pancreatitis is very indefinite, and varies greatly in different individuals. The patient will perhaps complain of vague epigastric pain, of occasional nausea and vomiting, and of "dyspepsia." There may be no more definite symptoms on which to base the diagnosis. The pain may be very slight, little more than discomfort, or there may be excruciating pain as in the paroxysms of gall-stone colic. Nausea and vomiting accompany the pain at its height, and in the intervals the patient may be free from symptoms. These do not appear to have any relation to food, although the patient usually diets himself in an effort to clear up his "indigestion." There is frequently jaundice, and in many cases the bowels are constipated. In severe cases constipation disappears and characteristic large pale fatty stools are passed. There are few physical signs, although occasionally an area of tenderness over the head of the pancreas is present.

Diagnosis.—The diagnosis of chronic pancreatitis is, from the clinical picture at least, one of considerable difficulty. It depends on the general symptoms of dyspepsia described above, but since the diagnosis from gastric dyspepsia on the one hand and from gall-stones on the other is a matter of much uncertainty, a number of tests have been devised depending on the functional disturbance of the gland, in order to supplement the clinician's evidence. Unfortunately, however, none of these tests has proved to be absolutely pathognomonic of pancreatic disease. The most serviceable are Cannidge's test (see URINE, EXAMINATION OF), an index of disturbed carbohydrate metabolism which may or may not be due to pancreatic disease; Læwi's adrenalin test, which is useful when there is a disturbed equilibrium between the glands of internal secretion; and the demonstration of a high diastase content in the urine (see Renal Efficiency Tests, under NEPHRITIS, p. 340) in pancreatic diseases. In Læwi's test, two drops of 1 : 1000 adrenalin are placed in one eye, and two drops of water in the other eye as a control. The pupils are examined after twenty minutes. In a large proportion of patients suffering from pancreatic disease there is dilatation of the pupil after adrenalin. If possible, the test should be repeated or the other eye, after the effect on the first has passed off.

A further useful test is the determination of the glucose tolerance of the patient. Nor

mally the greatest average amount of glucose which can be ingested at one time without producing glycosuria is 150 grm. In cases of pancreatic disorder the glucose "threshold" is often considerably lowered. In carrying out this test care should be taken to give small amounts (10-50 grm.) of glucose and to raise the quantity gradually until the threshold is ascertained. In giving an excessive dose much above the patient's threshold there is some danger that permanent glycosuria may be established.

In relying upon chemical tests, it is as well to carry out all of those named: a positive result in at least two is strong evidence of pancreatic involvement. Recently, Einhorn has claimed good diagnostic results with the use of his duodenal tube. The tube, with a heavy metal capsule, is swallowed and allowed to pass by peristalsis from the stomach to the duodenum. The fluid in the duodenum is drawn off by means of a syringe and tested for pancreatic ferments. Their absence he claims as a valuable diagnostic sign. There are, however, difficulties in the use of the tube which detract somewhat from the value of the test.

Prognosis.—The prognosis in cases of chronic pancreatitis depends on a correct diagnosis and early surgical treatment. If the disease is left to pursue its course, the infection spreads, gradually involving more and more of the gland, and resulting in a serious and progressive derangement of function. The progress is slow, since the gastric and intestinal juices to some extent perform the duties of the pancreatic juice as this ceases to function, but a stage is reached when glycosuria may supervene, leading to diabetes and coma, and fat-metabolism is seriously impaired, as evidenced by the bulky whitish stools, which are greasy from the presence of unsplit fat. The patient becomes thin and ill nourished, and a secondary infection often supervenes owing to his lack of resistance. With surgical treatment and drainage, however, the case will in all probability clear up, and the longer the drainage is continued the less is the danger of recurrence.

Treatment.—The treatment of chronic pancreatitis is surgical. Administration of pancreatin by the mouth is merely palliative; it may relieve the symptoms temporarily, but does not touch the root of the trouble. The procedure is to expose the pancreas and gall-bladder and insert a drainage-tube into the

bile-duct. The keynote of treatment is not the drainage of bile, but the reduction of pressure in the biliary system. The drainage provides a safety-valve against the rise of pressure in the pancreatic duct. Removal of the gall-bladder is attended with successful results, and the most satisfactory after-histories are found in those cases in which drainage has been kept up for at least three weeks, and preferably for longer. When drainage is only carried out for a few days there is considerable risk of a recurrence of the disease. After operation the patient should be kept on a bland anti-diabetic diet, so as to make as little call as possible on the pancreas, and the administration of pancreatin by the mouth is often useful.

C. M. WILSON.

PANOPHTHALMITIS.—An acute septic infection of the interior of the eyeball.

Etiology. The affection is usually caused by infection from without, and most commonly by a perforating injury, with or without the retention of a foreign body. It may also happen after intra-ocular operations, such as cataract extraction, or from the perforation of a septic ulcer. In eyes with cystoid or fistulous scars (e.g. after the operation of scleral trephining for glaucoma) micro-organisms occasionally pass through the epithelium and cause infection. Panophthalmitis occurs rarely as a metastatic condition in pyæmia, cerebro-spinal meningitis, etc., but the process is not then so acute, and the eye can often be retained.

Symptomatology.—In a case due to a perforating injury the wound, in twenty-four to thirty-six hours, takes on an inflamed appearance; if it is in the cornea, for example, the surrounding area becomes hazy, the wound edges become swollen, grey, and finally yellow; the iris is congested and the pupil does not respond well to atropine; lymph, at first grey, then yellow, appears in the pupil, and soon hypopyon is well established. Infection passes backwards into the vitreous, which becomes converted into pus. The intra-ocular tension rises if the wound has closed.

Edema of the lids occurs on the third day, and the conjunctiva and sclera become intensely inflamed. (Edema into Tenon's capsule and the orbital tissues causes proptosis and sometimes much limitation of the ocular movements. Pain is generally severe, and is associated with headache and pyrexia.

Cases with retention of a foreign body may show pus in the vitreous as the first sign, the

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micro-organisms having infected not the wound, but the deeper parts of the eye. In these the pus is visible through the pupil, appearing as a yellow mass behind the lens. Otherwise the symptoms are the same, and the anterior chamber soon becomes infected.

Similarly, in metastatic cases the infection is first in the vitreous cavity: some of these run their course with but little congestion outside the globe. They are due to septic emboli lodging in the retinal or choroidal vessels, and may be bilateral.

The **prognosis** is usually hopeless as regards vision, and the eyes generally have to be excised. Milder cases in which panophthalmitis threatens but does not become established are met with.

Treatment.—In the early stages of infection of a wound, whether operative or accidental, it is worth while to attempt disinfection. The wound should be touched with pure carbolic acid or with the cautery, and the anterior chamber may be washed out with weak hydrogen peroxide or acriflavine solution. The non-operative measures will include hot bathings, hot fomentations, and atropine.

Such treatment occasionally stops the process if the vitreous is not involved, but often it progresses and pain is only relieved by evacuation of the pus. This may be done by excision of the globe, but there is some danger of setting up meningitis by infecting the subdural space around the optic nerve, and it is perhaps safer either (a) to incise freely across the cornea and sclera, to allow of drainage of the abscess cavity, or (b) to perform the operation of *evisceration*. The conjunctiva is dissected up as for excision, the cornea and surrounding rim of sclera are removed by knife and scissors, the contents of the globe are scraped out, and the interior of the sclera is everted thoroughly, first with a sharp spoon and then with wool swabs, until it is certain that all the pigmented tissue has been removed. A gauze plug and drain are left *in situ*.

F. A. JULER.

PAPILLITIS (see OPTIC NEURITIS AND NEURO-RETINITIS).

PAPILLOEDEMA (see OPTIC NEURITIS AND NEURO-RETINITIS).

PAPILLOMA (see WARTS).

PAPILLOMATOUS CYSTS (see OVARIAN CYSTS).

PARAOENTESIS (see ASCITES; PLEURISY).

PARALYSIS AGITANS

PARALYSIS AGITANS.— Sometimes known as Parkinson's Disease, paralysis agitans is a disease of slow and insidious onset, characterized mainly by progressive weakness and tremor, starting usually in the early fifties. Rare under the age of 40, it is very occasionally met with between 20 and 30. Seldom is the disease itself hereditary, though often a history of nervous heredity can be traced.

Pathology and symptomatology.—Until lately the pathology of paralysis agitans was not based on any known morbid anatomy, though analogies have been traced with other conditions known to be due to chronic lesions of the basal nuclei. Further research has shown that paralysis agitans itself is always associated with, and probably the result of, chronic nuclear degeneration of the globus pallidus, the inner portion of the lenticular nucleus. It is frequently mistaken for neurasthenia in its onset; complaint may be made of nervousness, irritability, and often of pains referred to the neck and shoulders. One upper extremity is likely to develop weakness gradually, and it is noticed that the patient does not swing that limb in walking. Tremor of the hand when the limb is at rest during waking hours, which becomes worse under emotion or excitement, but ceases in voluntary movement, is next observed. At the same time, if the right hand is involved, the handwriting becomes smaller, and a tremulousness may be noticed especially if a fine pen is used. The hand may be held in the so-called interosseal position, the thumb adducted to the index finger, and rhythmic tremor suggestive of a pill-rolling or a cigarette-rolling movement may be observed, together with alternate flexion of the wrist. Immobility of the features is an important sign, the naso-labial fold being smoother on the affected side, and the general aspect is one of depression. The movements of the head and trunk are deliberate, and the gait tends to be more or less stiff; the leg on the side of the affected arm is the first to show weakness. In a fully developed case the gait is slow and shuffling, the knees are slightly bent, the body and head stooping forwards, the arms slightly flexed and held somewhat stiffly, while the fingers and wrists shake. As the patient moves forwards he may break into a run, the so-called "festination," or running after his centre of gravity. The tremor spreads to the lower limb, and the lower jaw and the tongue may also shake violently; salivation may be a prominent feature. The voice becomes high-

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pitched, monotonous, and difficult to understand, and in a longstanding case even speech may be lost. The intelligence, however, is unaltered, and it is advisable for a patient to keep at his daily work as long as he possibly can, as the progress of the disease, though slow, is sure and relentless. When the tremor of the limbs is violent the head may appear to shake when the patient is sitting, though this is only communicated vibration and not a true head tremor.

Rigidity of the muscles of the trunk, neck, and limb girdles is a characteristic feature of the disease, and it is this and the fixity of expression, the tottering gait, and the slowness of turning round that arrest attention and suggest the diagnosis. These signs are indeed much more characteristic than the tremor, which may be closely simulated by neurasthenic and also by postfebrile tremor. The rigidity and lack of facial expression, especially while talking, is sometimes known as the "Parkinsonian mask," a valuable diagnostic sign, for, if there is free mobility of the features and the arms swing freely in walking and in turning round, it is advisable to be chary of making a diagnosis of paralysis agitans, even though the tremor appears characteristic. It is true that occasionally tremor is the initial symptom, before facial immobility or trunk rigidity is noticeable, though it is much more common for the characteristic facies, the stiffness of gait, and the rigidity of the trunk and limbs to be well marked before any sign of tremor is to be observed; indeed, in some cases tremor is conspicuous by its absence throughout, or is only occasionally present. Though the appearance of the trunk and limbs suggests spasticity, yet the reflexes remain normal, the plantar responses being flexor in type. There is no muscular wasting, no anaesthesia or analgesia, the sphincters are unaffected, and the pupils, eye movements, and optic discs are normal. There are thus, so to speak, none of the ordinarily accepted signs of organic disease, and hysteria or neurasthenia is often wrongly diagnosed in the earlier stages of paralysis agitans.

The course of the disease varies considerably in different individuals. In some cases the progressive weakness of arm and leg on one side presents the picture of a slowly developing hemiplegia, and thus the diagnosis of cerebral tumour may be an error to avoid. Indeed, the hemiplegic gait with the pronounced limping on one leg is sometimes remarkable. In other

cases the disease runs a rapid course, and within twelve or eighteen months the full clinical picture of bilateral rigidity, tremor, stooping, with the typical facies and weak shuffling gait, may be established. Indeed, by this time the patient may be unable to walk alone across the room. Sometimes the disease is much slower in development, and it may be several years before the patient reaches the bath-chair or bedridden stage, the disease lasting perhaps twenty-five years. Those cases which show extreme rigidity but little or no tremor generally progress to a fatal termination in a period of fifteen to eighteen months. Occasionally the onset is sudden. Fright and prolonged mental strain appear to be undoubted agencies in the development of the disease, though it is more than doubtful whether they are more than mere accelerators of the symptoms. Many cases were observed to develop after the air-raids, to which they were ascribed, and I have seen another patient whose symptoms started immediately after the Silvertown explosion.

In the later stages contractures may become established. I have known the whole trunk rigidly flexed in a stooping position, and the patient was totally bedridden and speechless for seven years before death, the limbs and trunk being apparently quite immovable owing to rigidity. The immobility and rigidity was, however, not due to true contracture, because total relaxation took place after death. Some patients complain of a contraction of the toes within the boot while walking, due to interosseal flexion similar to that which occurs in the hand.

Treatment.—This is wholly unsatisfactory as regards any prospect of cure. Once the diagnosis is certain, the course of the disease and its end are equally certain. So long as the physical powers permit the patient to get about, or go to business, it is advisable that he should be urged to do so, though fatigue must be avoided, and worry and excitement should be eliminated as far as possible. Massage appears to give some relief for a time, though too much is not to be hoped from it; electrical treatment, I find, is wholly useless. The tremor and salivation may be kept in check, and the general nervousness, a feature in some cases of the disease, allayed by the persistent administration of hyoscine in doses of $\frac{1}{100}$ to $\frac{1}{120}$ gr. three times a day. With this may be advantageously combined a small quantity of sodium bromide and dilute hydro.

PARAMYOCLONUS MULTIPLEX

bromic acid. Often the patients feel great benefit from the hyoscine mixture and find that they cannot do without it, but gradually the dose has to be increased; a small dose should always be tried at first.

WILFRED HARRIS.

PARALYSIS, BELL'S (*see* FACIAL PALSY).

PARALYSIS, BROWN-SÉQUARD (*see* SPINAL CORD, LOCAL LESIONS OF).

PARALYSIS, BULBAR (*see* BULBAR PARALYSIS).

PARALYSIS, DIVER'S (*see* CAISSON DISEASE).

PARALYSIS, ERB'S (*see* SPINAL NERVES, LESIONS OF).

PARALYSIS, FACIAL (*see* FACIAL PALSY).

PARALYSIS, FAMILY PERIODIC (*see* PERIODIC PARALYSIS, FAMILY).

PARALYSIS, INFANTILE (*see* POLIO-MYELITIS, ACUTE).

PARALYSIS, KLUMPKE'S (*see* SPINAL NERVES, LESIONS OF).

PARALYSIS, LANDRY'S (*see* LANDRY'S PARALYSIS).

PARALYSIS, OCULAR (*see* OPHTHALMO-PLEGIA).

PARALYSIS OF PALATE (*see* PALATE, PARALYSIS OF).

PARALYSIS OF VOCAL CORDS (*see* VOCAL CORDS, PARALYSIS OF).

PARALYSIS, TRIGEMINAL (*see* TRIGEMINAL NERVE, PARALYSIS OF).

PARAMETRITIS (*see* PELVIC CELLULITIS).

PARAMNESIA (*see* MEMORY, DISTURBANCES OF).

PARAMYOCLONUS MULTIPLEX.—A disease characterized by clonic spasms in the muscles of the trunk and limbs. The spasms vary in strength; they may be insufficient to produce movement of the limbs, or they may jerk the patient about. In frequency they vary from ten to sixty per minute. They are increased by emotion, diminish on voluntary movement, and cease entirely during sleep. The muscles most often involved are the biceps, supinator longus, and quadriceps femoris.

PARANOIA

A special familial form of the disease described by Unverricht is associated with epilepsy, and shows a distinct tendency to dementia.

Many cases with involuntary movements which have been included in paramyoclonus have probably been hysterical.

Prognosis as to recovery is bad. Several cases which have been reported as cured—chiefly by electricity—were probably hysterical. Sedatives such as potassium bromide, chloral, and morphia will allay the spasms. In certain cases electricity and psychotherapy have had good effects. Such treatment should be tried even in cases presumably not hysterical.

F. C. PURSER.

PARANOIA.—A chronic mental disease characterized by the formation of a fixed delusion or a series of fixed delusions, which dominates the mental life of the patient. For practical purposes it is synonymous with the chronic delusional insanity or the monomania of the older writers.

Etiology.—Paranoia is a rare disease, and occurs more frequently in males than in females. Hereditary predisposition is a prominent feature. Some of the most typical cases are to be found in patients who have been addicted to alcohol. No characteristic abnormalities have been observed post mortem.

Symptomatology.—The disease is of so gradual a growth that years may elapse between its first appearance and a recognition of the necessity for medical advice. If a complete history can be obtained it will be found in most cases that the patient was always peculiar in some respect. In boyhood, instead of mixing with his fellows and participating in their games, he was moody and seclusive. Gradually a false relation with the environment is developed, and matters which to ordinary people would be trivial are construed as of special importance. Hallucinations, chiefly auditory, lend support to the false ideas, and thus slowly a delusional system of persecution is built up. The delusions of persecution may persist indefinitely, or may give place to ideas of grandeur, the reasoning being: "If I am subjected to so great annoyance, it must be because I am some great person." Paranoiacs may thus be classified under two main headings—(1) the persecuted, (2) the exalted. Numerous sub-varieties, such as the litigious, the religious, and the sexual, can be formed, but they serve no useful purpose.

The emotions, except when coloured by the delusions, and the will, except when influenced by them, are little affected. Outside the sphere of the delusional system, little may appear to the ordinary observer to be amiss. The memory is unimpaired, reasoning on other subjects may be intelligent, the patient is neither gloomy nor exuberant, and conducts himself naturally. Of course, if the imaginary persecution is severe he is the reverse of happy under it and may turn upon his supposed persecutors and proceed to extremes, even to murder.

Course and prognosis.—The course of the disease is slow, and the prognosis as regards mental recovery bad, although in some few cases, after many years, the delusions gradually fade and the patient is discharged recovered, often to the surprise of his physician. There is little tendency to dementia. The disease is not dangerous to life, and there is no reason why a paranoiac should not live as long as though he were sane.

Diagnosis.—A full history is indispensable in making a diagnosis. In this the essential points are the gradual growth of the insane delusions present on examination. In the exalted type the possibility of *general paralysis* should be thought of, and a careful examination made to exclude physical signs of this disease. The duration of the disease will be of assistance, for though cases of general paralysis have been recorded as persisting for many years, these are very exceptional. The paranoid form of *dementia præcox* is not always easy to exclude. In dementia præcox emotional deterioration is present, and there are few cases which do not exhibit the characteristic mannerisms or stereotypies of this disease. Delusions occur in the chronic forms of *mania* and *melancholia*; they do not, however, constitute the most prominent feature of the illness. In mania there is general craziness, as shown in speech and behaviour, and in melancholia there is an extreme state of depression. The delusions in these diseases are not so stable and persistent as in paranoia.

Treatment.—The patient should be certified and sent to an institution. Among the relatives there is often the not unnatural feeling that if a man is able to write a rational letter, and in many respects to converse and behave as a sane person, it is a great hardship to intern him in a mental hospital. Still, in many cases of the persecutory type, though at first the delusions may be of vague general persecution, the time is apt to come when the

subject traces his troubles to the instigation of a particular person, upon whom he may commit violence. In other cases he becomes a general nuisance, and it is best for society that he be incarcerated. Strangely enough, many of these patients do not feel the deprivation of freedom so intensely as might be imagined. Once in the mental hospital, there is little that can be done in the way of cure beyond regulation of occupation and relaxation. Psycho-analysis is reported to have proved beneficial, but in the few cases in which it has been tried the correct diagnosis would appear to lean rather to the paranoic form of dementia præcox than to pure paranoia.

R. H. STEEN.

PARAPHIMOSIS.—In children this condition is due to the pulling backwards, behind the corona, of a prepuce with a narrow opening. Then the margin of the preputial orifice, acting as a constricting ring, causes congestion and swelling of the glans, which becomes too large for the patient to return it to the shelter of the foreskin. In adults paraphimosis is usually associated with some inflammatory condition of the prepuce or preputial sac (venereal sores, balanitis). On examination the glans is found to be enlarged and pointing slightly upwards compared with the hinder part of the penis. Behind the corona is a deep cleft, the posterior margin of which is formed by an oedematous ring of tissue, the swollen mucosa of the prepuce. Behind this again is a cleft with the constricting ring at the bottom. The fissure is bounded by another collar, composed in this case of swollen skin. There is a good deal of associated pain, and if the condition is not relieved the constricting ring on the dorsum ulcerates. It is said that in some cases extensive sloughing of the prepuce and glans results; if this is so it must be exceedingly rare, for relief is usually obtained spontaneously by the ulceration. This, however, should never be permitted; it is accompanied by much septic infection and leaves a large raw area on the dorsum.

Treatment.—An early case can often be reduced by first gently pressing the oedematous glans to reduce its size and then dragging the foreskin forward again. In all but early cases an anæsthetic must be administered before attempting this manoeuvre, firstly because it is painful, and secondly to enable an incision to be made should it prove unsuccessful. When incision becomes necessary it should be

made on the dorsum at the bottom of the groove containing the constricting ring. The oedema of the prepuce can be then expressed and the foreskin drawn forward. Circumcision should be reserved for a later occasion. In adults, when the paraphimosis is of inflammatory origin, after reduction the whole foreskin should be slit up along the dorsum. This will allow of treatment of the underlying causal lesion.

C. A. PANNETT.

PARAPLEGIA, ATAXIC (see ATAXIC PARAPLEGIA).

PARAPLEGIA, COMPRESSION (see PARAPLEGIA, SPASTIC).

PARAPLEGIA, SENILE.—As old age comes on, the legs grow feebler and gait assumes a character that is easily recognized: the steps become shorter, the movements of the whole body stiffer and more rigid, and the limbs lose the flexibility and elasticity that distinguish them in the locomotion of healthy youth. This condition is not usually due to, or associated with, gross organic changes in either the nervous or the muscular system.

A true paraplegia does, however, occasionally develop in old age, which cannot be attributed to any of the ordinary causes that produce this condition in earlier life. Its first symptom is generally a feeling of stiffness and weakness of the legs and a tendency to tire rapidly on exercise. This gradually increases till walking becomes affected; then the legs move slowly and feebly, owing partly to their increasing weakness and partly to the rigidity of the muscles, the steps become short, the toes cling to the ground and catch on every obstacle or irregularity on it, and the maintenance of equilibrium grows more difficult. The condition rarely progresses beyond this stage.

Examination reveals a spastic weakness of the legs of moderate degree, which is usually symmetrical but may be more marked in one limb than in the other. There is rarely any demonstrable weakness of the trunk muscles, and in the arms there is seldom more than the tremor and feebleness that accompany old age. The knee- and ankle-jerks are generally brisk or exaggerated, the plantar reflexes are extensor, and the abdominal reflexes absent or diminished. As a rule, sensory disturbances are absent and the sphincters are rarely affected.

The **pathology** of senile paraplegia is often uncertain, but the most common cause is

the presence of multiple small lesions of vascular origin in the cord, and of small foci of sclerosis around the arterio-sclerotic vessels. Disseminated vascular lesions in the brain or brain-stem may, however, produce the same symptoms. Lhermitte has shown that in certain cases the symptoms are due to fibrotic changes and contractures in the muscles rather than to nervous lesions, but in these cases there are no changes in the reflexes, or at most a difficulty in eliciting the knee- and ankle-jerks.

Treatment.—Rest, with short periods of exercise, is essential. Massage is of great benefit, as it reduces the rigidity of the muscles and makes voluntary movement more easy. Drugs are of little value, but iodide in small doses may be tried.

GORDON HOLMES.

PARAPLEGIA, SPASTIC.—The term paraplegia is generally used to signify paralysis or loss of power of the lower limbs, and of a part or of the whole of the trunk; it is, in fact, applied to impairment of the motor and sensory functions of the legs, regardless of its nature or origin. Paraplegia is consequently not a disease, and is no more a clinical entity than is a cough; it is merely a symptom common to a large number of different nervous affections which impair the functions of the lower limbs.

When we meet with a case of weakness or paralysis of the legs we have therefore to consider the cause to which it may be due. It may be, in the first place, produced by injury or disease of the peripheral nerves that innervate the muscles of these limbs, or by affections of the spinal roots, particularly in the cauda equina where they are all grouped together, or of the grey matter of the cord, with which these nerves are in connexion. Weakness of the legs is, for instance, commonly the most obvious symptom of polyneuritis or a generalized peripheral neuritis, or it may result from poliomyelitis. But as these affections are rarely confined to the lower limbs and the lower part of the trunk, the term paraplegia is seldom appropriate.

Then any disease of the spinal cord below the cervical enlargement, whether it originates in the cord or involves it secondarily, may produce the clinical picture of a paraplegia; and in rare cases cerebral lesions may be the origin of it. As in these cases the upper, or cortico-spinal, neurones are involved, the disease when fully developed is spastic—that is, the limbs

PARAPLEGIA, SPASTIC

are rigid or hypertonic, the muscles do not waste, and the tendon-jerks are exaggerated. Finally, loss of power in the legs may be a manifestation of hysteria—hysterical paraplegia.

We must consider to which of these classes each case belongs, and then its cause. Hysterical loss of power and the paralysis due to peripheral nerve injuries and diseases have been discussed in other articles, so that here we have to deal only with those forms due to organic affections of the central nervous system, and as these produce, as a rule, a spastic weakness with related changes in the reflexes, most of them may be included under the clinical term spastic paraplegia.

1. SPASTIC PARAPLEGIA OF SPINAL ORIGIN.

—Spastic paralysis results from any disease that affects the functions of the upper motor neurones subserving the voluntary movements of the lower limbs. There are frequently sensory changes too, as the sensory conducting fibres of the cord are often involved, and the sphincters rarely escape if the disease is at all severe.

Such spinal disease may be local—that is, limited to a small portion of the cord; or it may be diffuse or irregularly distributed throughout it; or systemic—that is, a degeneration of certain tracts of fibres only.

(1) **Spastic paraplegia due to local spinal lesions.**—Here the primary disease may be either intramedullary—that is, it may affect the cord itself primarily—or extramedullary, originating outside the cord and disturbing its functions by compression or secondary invasion. But in both cases the symptoms are due to the same cause, viz. to interference with the functions of the motor spinal fibres.

A *local intramedullary lesion* may be due to many different causes which can be distinguished only by the clinical forms they take, or by the conditions with which they are associated. It may be the result of a trauma that produces either a direct injury of the cord or a softening or a hæmorrhage into it, or may be due to a focus of inflammation, myelitis, or softening secondary to vascular occlusion, or to a tumour or abscess that develops within the cord. The symptoms, pathology, and treatment of these conditions are dealt with in other articles, so that here we have only to discuss their clinical features in so far as they can help us in the differential diagnosis of paraplegia.

It will be noticed that, with the exception

of tumours, all these intramedullary spinal lesions set in acutely or suddenly; trauma usually produces its effects at once, and hæmorrhages and softenings destroy the tissues within a short time; myelitis, too, is generally an acute condition. Consequently, the first striking feature of most of these local intramedullary lesions is the acute onset of the symptoms. The loss of power is sometimes quite sudden, or apparently so, and in other cases it becomes complete or reaches its full development within a few days. There may be prodromata, as transient or recurring numbness or weakness of the legs, or temporary retention of urine, but these warnings are not common.

A second feature of intramedullary lesions is the absence of pain. It is a rule to which there are few exceptions that disease limited to the grey or white matter of the cord does not produce pain which is either referred peripheralwards, or locally to the spine; even the severest inflammation of the cord may run an absolutely painless course unless the extramedullary portions of the sensory roots are involved in it.

In the third place, as the onset is generally acute or sudden, the paralysis is at first flaccid—that is, the muscles are relaxed and toneless, the knee- and ankle-jerks are absent or depressed for some days at least, and during this stage it may be impossible to elicit any reflex by stimulating the soles. But if the transverse section of the cord is not completely destroyed the paralysis gradually becomes spastic about three weeks after the onset. The first sign of the developing spasticity is usually an exaggeration or an increasing briskness of the knee- and ankle-jerks and the appearance of ankle-clonus. Then the muscles grow less flabby, spasms may occur, and passive movement of the limbs encounters more resistance. Thus we have the characteristic picture of a spastic paraplegia.

Compression paraplegia is due to conditions which develop outside the spinal cord and interfere with its functions by compressing it within the vertebral canal, or by producing secondary changes within it. The causes of compression may be classified according to the structures in which the disease commences.

i. Tumours growing from the *vertebral canal* easily compress the cord; they may be primary tumours of the bone, as osteomata or sarcomata, or secondary deposits in it, as malignant metastases from other organs. But the vertebral lesion that most commonly produces

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paraplegia is tuberculous caries ; the cord may be compressed by a tuberculous abscess that points into the canal, or the acute angular deformity that often develops may injure it by stretching or by constricting it within the narrowed canal.

ii. Or the compressing lesion may develop in the *membranes* or in the *extramedullary* portions of the *spinal roots*. It may be a tumour : simple fibromata attached to either the roots or the membranes are not uncommon ; endotheliomata and sarcomata may grow from the meninges and malignant metastases occur in them. Occasionally hydatid cysts are found here (*see SPINAL CORD, TUMOURS* of).

iii. Finally, the disease may be *primarily meningeal*. Syphilitic gummatous meningitis is the most common example of this type. Later, the fibrosis of the thickened meninges, whether the disease is syphilitic, tuberculous, or simple inflammatory, may lead to structural changes in the cord by compressing it, or by interfering with its blood and lymph supply. One important and relatively common cause of compression is the formation of cysts or loculated collections of cerebro-spinal fluid in the membranes. The pathology of this condition is still obscure, but in many cases it is a sequel to a mild attack of meningitis or to an injury of the spine. The cystic collections lie in the subarachnoid space and are contained within thickened and opaque portions of the arachnoid.

Several features distinguish paraplegia due to compression from that produced by primary intramedullary disease. In the first place, as the tumours, abscesses, cysts, and meningeal lesions that may compress the cord develop more or less gradually, the onset of the nervous symptoms is usually much slower than when the original disease is intraspinal. But this rule has exceptions ; a gummatous meningitis often causes rapid loss of power, and in spinal caries the paralysis occasionally develops more or less suddenly, either owing to the collapse of a vertebra or to the rupture of an abscess through the anterior common ligament into the vertebral canal.

In the second place, lesions that compress the cord involve almost always the *spinal roots* on its surface, or if they originate in the bone they may nip them in the intervertebral foramina. Damage of the ventral roots may cause local atrophies of one or more muscles, but this is not a common or characteristic symptom of extramedullary disease. Com-

pression or invasion of the dorsal roots, on the other hand, produces very frequently pain that is referred to their cutaneous distributions. In the limbs it may be limited to the areas of one or more roots, and on the trunk it may appear as a girdle pain. The pain is generally continuous and independent of any obvious cause, or it may be excited or aggravated by movements of the spine, especially when this is the site of the disease. It has most frequently a sharp lancinating or burning character, or it may be described as merely an unpleasant numbness, constriction, or tingling.

A third feature of compression paraplegia is that, as it develops slowly, a stage of flaccid paresis does not precede the rigidity ; as a rule the muscles of the limbs become spastic as they lose power, unless it be that the lumbosacral segments and the roots that take origin from them are involved. Finally, sphincter disturbances are usually less pronounced in relation to the amount of voluntary paresis than they are in paraplegia due to intramedullary lesions.

But despite these signs, on which we usually rely, it must be confessed that we are frequently in doubt as to whether the disease is primarily intra- or extramedullary.

(2) **Paraplegia due to diffuse spinal lesions.**—Several conditions produce diffuse or scattered spinal lesions rather than local damage to the cord, and, as these too involve the pyramidal tracts, the clinical state of spastic paraplegia results. The most common of these conditions are disseminated sclerosis and spinal syphilis ; their symptoms and diagnosis have been dealt with in the articles on **DISSEMINATED SCLEROSIS** and **CEREBRO-SPINAL SYPHILIS**. Irregular patches of degeneration and secondary sclerosis are occasionally due to diffuse vascular lesions, particularly in senile arterio-sclerosis (*see PARAPLEGIA, SENILE*), or are a result of a spinal meningitis.

The clinical symptoms of the paraplegia produced by diffuse lesions have many points in common. In the first place, sensation is generally affected as well as motility, though its disturbances are usually slight and may be merely subjective. In disseminated sclerosis, for instance, the patient complains almost invariably of numbness or other paræsthesiæ, and careful examination usually reveals alterations in certain forms of sensation. These are more common when the lesions are of syphilitic or meningeal origin.

Secondly, the symptoms are most pronounced

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in the caudal portions of the body and in the distal segments of the limbs, since the pyramidal fibres that innervate them are involved during their downward course in the cord by a succession of foci, while those that terminate in the higher segments are exposed to less damage.

In the third place, it is generally impossible to define accurately the upper limits of the disease; a patient may complain of weakness of his legs only, but absence of the abdominal reflexes indicates that the lower part of the dorsal cord is also affected, and exaggeration of the arm-jerks may show that the upper limbs have not escaped. Or paræsthesiæ, slight sensory disturbances, tremor, or a mild defect in co-ordination often reveal disease at a higher level of the cord than casual observation would suspect.

Finally, an accurate history frequently shows that in these diffuse conditions the symptoms develop by a series of successive attacks, and not gradually and uniformly as is usual when they are due to local spinal lesions or to systemic degenerations.

(3) **Paraplegia due to systemic degenerations of the pyramidal tracts.**—In the past a large proportion of cases of spastic paraplegia were attributed to a primary lateral sclerosis, but pathological as well as clinical investigations have shown that an isolated primary degeneration of the pyramidal tracts, as this term implies, is very rare. That it does occur there can be no doubt, but it is certainly one of the least common causes of spastic weakness of the legs. It is most commonly seen in children, often as a familial or an hereditary disease, and is one of the conditions included in Little's disease, or spastic diplegia. Strümpell has described a familial form that occurs in later life, commencing generally between 20 and 30 years of age, but in some of his cases other spinal tracts in addition to the pyramidal bundles were affected. More frequently the symptoms of primary lateral sclerosis are due to local or diffuse lesions of the nervous system, as disseminated sclerosis, which is certainly its most common cause, or to combined or multiple degenerations. Amyotrophic lateral sclerosis must never be forgotten as a possible cause, especially when examination reveals no sensory disturbances, ataxia, or other symptoms, for it happens not infrequently in this disease that the degeneration of the pyramidal fibres precedes for a long time the affection of the grey matter of the ventral horns that produces the characteristic wasting of the muscles.

The chief clinical features of primary lateral sclerosis are its very slowly progressive course, the absence of objective and subjective sensory disturbances, the predominance of spasticity over weakness, and the escape of the sphincters till late in the disease. It can be distinguished from local spinal lesions by the fact that a definite upper level to the symptoms cannot be detected, and from diffuse lesions of the cord by the absence of sensory symptoms and by its slow and gradual progress.

2. PARAPLEGIA DUE TO CEREBRAL LESIONS.

—As lesions of the brain usually produce hemiplegia, or at least a greater weakness of one side of the body than of the other, and as the rule is that the paralysis is more marked in the arm than in the leg, the clinical picture of paraplegia rarely results. But affection of both hemispheres at the upper ends of the fissures of Rolando, or damage of the fibres that spring from them, may produce a spastic paresis of the lower limbs and of the lower part of the body. Thus, thrombosis of the superior longitudinal sinus, or a wound or injury of the skull in this region, may paralyse the legs only. Or this may be the result of a double partial hemiplegia, due to two separate lesions or to multiple softenings, such as occurs in arterio-sclerosis. Lesions in the brain-stem, as tumours or softenings in the midbrain, pons, or medulla, may involve the pyramidal tracts on both sides, but since the fibres concerned in innervation of the lower limbs do not run separate from those of other functions the result is generally a tetraplegia rather than a paraplegia.

The upper motor neurones that innervate the legs are also liable to suffer in hydrocephalus, especially when it develops in early life. In general paralysis of the insane, weakness of the legs is sometimes a prominent symptom and may be so great as to assume the form of a spastic paraplegia; and in infantile cerebral diplegia, no matter to what cause it is due, the legs may be more affected than the arms.

There is rarely much difficulty in diagnosing weakness of the legs due to cerebral disease. In the first place, a careful examination generally shows that the arms are also affected, and frequently the bulbar mechanisms too, producing dysarthria, dysphagia, and the symptoms of a pseudo-bulbar palsy. Then sensory symptoms are usually absent, and there may be other signs that can be explained only by cerebral disturbances.

PARAPLEGIA, SPASTIC

Symptomatology of spastic paraplegia.—

We can now discuss the symptoms of spastic paraplegia regardless of its origin.

An accurate history of the mode of onset is the first, and often the most important, point in investigating any case of weakness of the legs, for while examination informs us of the present state of the patient, it is mainly on the history of its development that we rely in the diagnosis of its cause. In no other branch of medicine is a complete and accurate history so essential.

It is advisable to commence by the examination of the *motor system*. The muscles are, as a rule, well developed and not wasted, though they may appear smaller than normal if the paralysis is of long standing. Their tone is, however, always increased, and the rigid limbs offer a definite resistance to passive movement. The extensors are usually more spastic than the flexors, especially those of the knees and ankles, and the adductors and internal rotators than their opponents. Consequently the limbs generally lie in bed extended, and are often firmly adducted and rotated inwards. A flexor rigidity occurs only in the later stages, or as a sequel to a severe acute lesion. Tonic spasms are common even in early and mild cases; the limbs may either "shoot out" suddenly and remain for a time tonically extended, or they may be "drawn up" by flexor spasms. Contractures or organic shortening of the muscles frequently occur, especially in bedridden patients; the calf muscles, the adductors of the thighs, and the hamstrings are the most likely to shorten.

The degree of paresis is often much less than the disability of the limbs would suggest, as the rigidity impedes and restricts the movements that are possible. The weakness is usually greater in the distal segments, particularly in the toes and the dorsiflexors of the ankles. There are several phenomena which are more or less characteristic of the spastic limbs. When the patient attempts to raise himself from the supine position the legs often rise from the couch on which he lies, and as this brings the centre of gravity above the pelvis the attempt is difficult or unsuccessful—*Babinski's flexion combinée*. And in depressing one leg against resistance the other is often raised by involuntary contraction of the flexors of the hip. Again, on flexing the knee passively the tibialis anticus muscle contracts—the *tibialis sign* of Strümpell.

The gait has the typical spastic character;

the earliest trouble in slowly progressive cases is a feeling of stiffness and awkwardness of the legs and a tendency to tire quickly. At this stage walking on the flat may not be obviously abnormal, but actions that require more delicate movements, such as dancing and climbing, are difficult or impossible. The next symptom is a difficulty in raising the feet properly and a tendency to trip over obstacles and on rough ground. Then, as the legs become weaker and stiffer the steps grow shorter, the knees are not bent naturally, and the base often becomes narrower owing to the adductor spasm that keeps the knees together. This makes the maintenance of equilibrium more difficult, but there is no real ataxia of locomotion unless there are also sensory disturbances.

The muscles of the trunk also share in the paresis if the lesion lies higher in the cord than the tenth dorsal segment; its mild degrees may be difficult to demonstrate, but, if it is marked, the abdominal muscles or some portion of them do not contract firmly as the patient tries to sit up or to bend his head forwards against resistance; if only their lower portions are weak, the umbilicus may rise as the weak portions are stretched, while the upper part that contracts shortens. In severe paresis coughing may be much affected. When the intercostals are weak the lower ribs are not properly raised in inspiration, and their failure to contract may be felt if the observer's fingers are placed in the intercostal spaces.

Sensory disturbances are variable; they are absent when there is merely a systemic degeneration of the pyramidal tracts and when lesions are limited to them, usually slight in diffuse affections as disseminated sclerosis and spinal syphilis, and even in local lesions the loss is often minimal or affects certain qualities of sensation only. But in many cases sensation is equally affected with motion.

The changes in the reflexes are very important in diagnosis. The knee-jerks are invariably exaggerated and frequently clonic, and a definite patellar clonus may often be obtained. When there is considerable spasticity a tap on the patellar tendon or on the inner condyle of the femur often produces adduction and inward rotation of the thigh too. The ankle-jerks are similarly exaggerated, and ankle-clonus can usually be elicited. Stimulation of the sole always evokes extension of the great toe and spreading of the other toes (*Babinski's sign*), and stroking along the inner margin of the tibia often causes dorsiflexion of the foot

and of the great toe (*Oppenheim's sign*). The abdominal reflexes are absent if the lesion extends as high as the middle of the dorsal cord, and the cremasteric reflexes are usually abolished too. The state of the sphincters and organic reflexes is very variable. Frequently the patient experiences no difficulty in micturition even though there is considerable paresis of the legs, especially when this is due to systemic degeneration of the motor tracts. The general rule is that as the weakness progresses there occurs first a slowness in starting the flow of urine; or there may be urgency,—that is, when the desire to micturate comes the patient cannot restrain evacuation for more than a moment or two. The difficulty in starting micturition increases as the paralysis develops, till there may be retention, or retention with overflow-incontinence. Reflex incontinence, or involuntary evacuation of the bladder when the intravesical pressure rises to a certain height, occurs in severe paraplegia. Constipation, with a tendency to incontinence of fæces when diarrhoea occurs, is the usual form of rectal disturbance. Spastic paraplegia generally entails sexual impotence in man. Priapism is common in severe degrees of paralysis.

If the lesion to which the paraplegia is due increases until it produces a severe or total transverse section of the cord, the spasticity disappears and gives place to a flaccid paralysis with gross sensory disturbances, wasting of the muscles, trophic changes, and diminution or absence of the tendon reflexes.

A careful investigation of the symptoms and physical signs of paraplegia enables us to diagnose the nature of the disease and, if it be local, its position in the cord. The localization of spinal lesions is considered in another article (see SPINAL CORD, LOCAL LESIONS OF), and the principles of diagnosis are discussed in the opening sections of this article.

Treatment.—The treatment must depend on the nature of the lesion, but there are certain principles which must be borne in mind, no matter what the disease may be. In the first place, the development of contractures must be prevented by ensuring that the paralysed limbs are not allowed to lie constantly in the same position, and by the regular employment of passive movements and massage. Splints and simple apparatus are sometimes necessary to correct wrong postures. Electrical treatment and all peripheral stimuli that tend to excite spasm should be avoided. In the next place, the disturbances of gait should

be carefully analysed, as it is frequently possible for the patient to walk much better when errors are corrected either by re-education or by surgical or mechanical means. Tenotomy may be required if contractures have developed, and the functional value of the limbs can occasionally be improved by tendon transplantations. When the movements of the limbs are relatively strong and the disability is due largely to rigidity, gait can often be improved by section of the dorsal spinal roots (*Förster's operation*), but this should be attempted only when the disease is stationary.

The bladder symptoms require careful attention; by insisting that the bladder be emptied frequently, retention and its serious results can often be avoided. Incontinence necessitates aseptic precautions and the provision of a portable urinal.

GORDON HOLMES.

PARAPSORIASIS.—The name parapsoriasis has been applied by Brocq to a group of psoriasiform and lichen-like eruptions which do not conform to psoriasis, lichen planus, or any of the ordinary recognized types of skin disease, but have so many essential features in common as to warrant their inclusion under one heading. The eruptions in this group have been divided into three types—*parapsoriasis en gouttes*, in which the lesions are drop-like; *parapsoriasis lichenoïde*, in which they are arranged in a peculiar reticulate manner; and *parapsoriasis en plaques*, in which they form well-defined patches.

Symptomatology and histopathology.

—The essential lesion is a macule or maculopapule, round, oval, or irregular in outline, smooth or covered with a fine adherent scale, and varying in colour from fawn to pale red. By the spreading and coalescence of these initial lesions in different fashions the three named varieties are produced. The eruption is distributed chiefly on the trunk, arms, and legs, more rarely on the backs of the hands and dorsa of the feet, and is absent from the scalp and face. There are no subjective symptoms associated with it except slight itching when the skin is exposed to excessive heat, nor does it seem in any way to affect the general health. The evolution of the eruption is slow, its duration long, and it is extraordinarily resistant to treatment, local applications which would remove the lesions of psoriasis having little or no effect upon it.

The cause and true nature of parapsoriasis

PARATHYROID AFFECTIONS

are unknown. The histological features common to all the members of the group suggest that the primary changes occur in the blood-vessels and that the epidermal changes are secondary.

Diagnosis.—The diagnostic features are the slow evolution of the lesions, their chronicity, extraordinary resistance to local treatment, and the absence of subjective symptoms. The disease has to be distinguished chiefly from psoriasis, seborrhœic dermatitis, and lichen planus. From *psoriasis* it is differentiated by the lesions being less bright in colour, the scales not silvery, more adherent, and when scratched off not leaving a shiny film with bleeding-points, by its not affecting the sites usual for psoriasis, and being more resistant to treatment. From *seborrhœic dermatitis* it is distinguished by the scales being dry and white instead of yellowish and greasy, and by not occurring on the face and scalp, which are usually attacked by seborrhœa. From *lichen planus* the initial lesions may be diagnosed by being less prominent, not violaceous in tinge, never umbilicated, generally scaly, and unassociated with subjective symptoms.

Treatment.—The prognosis is unfavourable and neither local nor general treatment seems to have much effect, especially in the reticulate variety. In the guttate and patchy types, improvement may be obtained by removing the scales with soft soap and hot water and applying a strong ointment containing pyrogallol or salicylic acid. In isolated patches benefit may be derived from the X-rays employed as in psoriasis. Where the disease is widely distributed a mild resorcin or salicylic-acid ointment should be substituted, as the extensive use of stronger remedies is liable to set up an exfoliative dermatitis.

J. M. H. MACLEOD.

PARASYMPATHETIC SYSTEM (*see* VEGETATIVE (SYMPATHETIC AND PARASYMPATHETIC) SYSTEM).

PARATHYROID GLANDS, AFFECTIONS OF.—The rôle played by the parathyroid glands in the animal economy is little understood. The experiments of Gley and Vassale leave little doubt that the tetany which follows complete extirpation of the thyroid is really due to inclusion of the parathyroids, for if these be left no tetany occurs. As the result of their work the parathyroids have been carefully studied in cases of tetany in children,

PAROTID GLAND, TUMOURS OF

with very contradictory results; whilst the administration of these glands is only occasionally attended by benefit in that disease. McCallum and Voegtlin, by experiment, came to the conclusion that the parathyroid glands control calcium metabolism, their removal or inefficiency leading to a leakage of calcium by the bowel and consequently to hyperexcitability of nervous tissue. Of importance in this connexion is the low calcium content of the blood which occurs in tetany, and the good effect which attends the administration of calcium in that condition. The supposed connexion between parathyroid affection and paralysis agitans and myasthenia gravis is less well founded. (*See also* ORGANOTHERAPY.)

FREDERICK LANGMEAD.

PARATYPHOID INFECTIONS (*see* TYPHOID AND PARATYPHOID FEVERS).

PARENCHYMATOUS NEPHRITIS (*see* NEPHRITIS).

PARKINSON'S DISEASE (*see* PARALYSIS AGITANS).

PARONYCHIA (*see* WHITLOW).

PAROREXIA (*see* STOMACH, FUNCTIONAL DISORDERS OF; PICA).

PAROTID GLAND, TUMOURS OF.—Tumours of the parotid gland may be benign or malignant. The benign tumours comprise lipoma, chondroma, adenoma, and myxoma, while the malignant include sarcoma, carcinoma, and endothelioma. To enumerate the various neoplasms thus, however, is of little value unless the relative frequency of each type be given. For practical purposes there is only one common parotid tumour—the mixed tumour, which should be classed as an endothelioma. Carcinoma and sarcoma may occur, the former usually being of the scirrhus type.

Pathology.—The nature of the mixed parotid tumour was for a long time a debatable question among pathologists, but it is now generally regarded as an endothelioma, i.e. a form of sarcoma arising from the endothelium of a blood- or lymph-vessel. It is generally encapsuled, and, after attaining a size varying from that of a marble to that of an orange, may for many years undergo no change. Sooner or later it tends to grow and to undergo internal change. If the capsule is destroyed at any place the growth is prone to extend at that spot. The cells of the tumour may

PAROTITIS

undergo mucoid degeneration, whilst at other times a substance resembling cartilage is formed. The cartilage in parotid tumours probably has no connexion with Meckel's cartilage. Rarely, other forms of sarcoma may occur in the gland, but they need no special description. Carcinoma may be of the fungating variety, but is much more frequently of the atrophic scirrhous form.

Symptomatology and diagnosis.—A slow-growing, painless, and slightly mobile tumour developing in the parotid region, with the skin freely movable over it, can be diagnosed correctly as a mixed tumour in nine out of ten cases. It may gain a great size without causing any inconvenience other than the unsightliness. The facial nerve is very seldom implicated.

Atrophic carcinoma forms a hard swelling, at a certain stage becoming adherent to the skin and causing puckering. Glands at the angle of the jaw become enlarged. The facial nerve is generally implicated, and facial palsy results.

In well-developed cases there is no mistaking a mixed tumour, but in the early stages the small swelling may be mistaken for an *enlarged lymph-gland*. For the latter a cause can usually be found. *Actinomycosis* of the parotid appears as a large hard swelling, but ultimately sinuses form and the fungus can be obtained.

Advanced scirrhous-carcinoma presents a typical picture, but in early stages diagnosis may be difficult; the facial palsy is of great importance in differentiating. In doubtful cases a portion should be excised and examined microscopically. Caution is necessary not to mistake a diffuse parotid gland for a pathological condition.

Treatment.—The mixed tumour should be excised, along with its capsule. This can usually be done without injuring the facial nerve. Care must be taken to ligature securely any divided vessels.

The whole parotid gland is sometimes excised for carcinoma, but it is doubtful whether this is often possible or justifiable. Failing an attempt at excision, radiotherapy should be employed.

ZACHARY COPE.

PAROTITIS.—The parotid gland is bounded by firm bony and fascial structures, and any inflammation is especially painful owing to the tension produced. The branches of the facial nerve course forward through the gland. The parotid duct (Stenson's) crosses the cheek superficially to the masseter along a line drawn

from the tragus to midway between the columella of the nose and the red margin of the lip; the opening into the mouth is opposite the second upper molar tooth.

Etiology.—Parotitis is due to infection of the gland with micro-organisms. The bacteria may reach the gland by the blood-stream, by the excretory duct, or by penetrating injury.

Blood infection.—Epidemic parotitis (mumps, q.v.) is the best example, but in pyæmia abscesses may develop in the parotid.

Infection via Stenson's duct is the most common cause of the non-epidemic form of the disease. The mouth contains many pathogenic germs even in healthy subjects, and when pyorrhœa or carious teeth are present the possibility of infection is very great. In feverish patients the mouth becomes dry, sordes accumulates, and microbes multiply, thereby increasing the risk of parotid infection. If after an abdominal operation the surgeon adopts a policy of semi-starvation, and the hygiene of the mouth is not carefully attended to, parotitis may follow, for in the absence of the normal stimulus to parotid secretion the flow of saliva ceases and bacteria can gain an entrance to the duct.

Pathology.—The inflammation may be (a) severe enough to cause acute necrosis of the whole gland; (b) less severe, causing suppuration which may lead to discharge of pus into the mouth along the duct, or be opened by an external incision; (c) mild and non-suppurative. The virulence of the microbe and the resistance of the patient determine which shall occur. Staphylococci and streptococci are the usual organisms present.

Symptomatology.—Painful swelling of the gland, inability to open the mouth properly, and referred pain in the ear and along the course of the auriculo-temporal nerve, are the main symptoms. Redness of the overlying skin is a late symptom. Fluctuation is rarely felt. Fever is present, and in severe necrotic cases the toxæmic symptoms are extreme.

Diagnosis is usually easy, owing to the anatomical limitation of the pain and swelling. Pus oozing from Stenson's duct may confirm the diagnosis.

Prognosis.—Recovery is the rule, except in the severe necrotic cases occurring in debilitated subjects or in tropical climates.

Treatment.—Prophylactic hygiene of the mouth is essential. Thorough treatment of pyorrhœa and of dental caries, careful cleansing of the teeth and tongue in fevers and after

PAROXYSMAL TACHYCARDIA

abdominal or other operations, may prevent the onset of parotitis. When inflammation has begun, fomentations give relief to the pain. If from the local indications it is thought that an abscess has formed, it should be opened by a small external incision by Hilton's method; but if the symptoms of toxæmia are severe and necrosis of the gland is feared, free incisions should be made immediately. A large inverted T-shaped cut at the angle of the jaw allows any sloughs to come away, but an additional cut should be made just behind the auricle. After incision, fomentations should be applied and the wound treated antiseptically. The micro-organism should be cultured and an autogenous vaccine made and utilized.

ZACHARY COPE.

PAROTITIS, EPIDEMIC (*see* MUMPS).

PAROVARIAN CYSTS (*see* OVARIAN CYSTS).

PAROXYSMAL HÆMOGLOBINURIA (*see* URINE, EXAMINATION OF).

PAROXYSMAL TACHYCARDIA.—Sudden attacks of extreme acceleration of the heart which last from a few seconds to a few days and end as abruptly as they begin. The rapid beats constituting the paroxysm arise from an abnormal site in the myocardium and may be regarded as a succession of premature contractions.

For convenience, the paroxysmal forms of auricular fibrillation and flutter are discussed in the articles **AURICULAR FIBRILLATION** and **AURICULAR FLUTTER**.

Etiology.—The causation of paroxysmal tachycardia is unknown, and but a small proportion of the cases give a history of acute rheumatism. It is not a common disease; among 7,500 soldiers with various ailments referred to the heart, I saw only 10 cases of paroxysmal tachycardia, and this although the incidence is greatest in young adults and in males.

Pathology.—Post-mortem examinations have not revealed a lesion characteristic of this disorder of rhythm. In a few rheumatic cases, mitral stenosis or myocardial disease has been found in association, and in some syphilitic hearts occlusion of a coronary artery or one of its branches.

Experimentally, it is possible to induce a paroxysm by repetitive stimuli applied to the heart-muscle, or by ligation of a branch of the

coronary artery. The new site of impulse-formation supersedes the normal pacemaker of the heart (the sino-auricular node), chiefly by reason of its excessive rate of production, and originates a tachycardia of unalterable rate which is outside nervous control.

Symptomatology. (1) **Between attacks.**—The majority of patients feel well in the intervals between the attacks. A few are shorter-winded than healthy people, and complain of everyday symptoms such as dizziness, slight palpitation, and exhaustion on effort. It is the exception to find any physical signs of cardio-vascular disease. If found they are generally referable to some antecedent disease, usually acute rheumatism or syphilis. The only sign at all common in uncomplicated cases is an intermission of the pulse, due to isolated premature contractions which an electrocardiogram may refer to the same irritable focus in the heart as that from which arise the constituent beats of a paroxysm.

(2) **During attacks.**—Without warning and without apparent cause the patient suddenly becomes aware of a rapid action of the heart and a throbbing in the neck. He may feel a "rising" to the throat, and a dizziness when he stands. On walking he finds himself more breathless and more easily tired than usual. One accustomed to previous attacks will merely sit or lie down and wait for the attack to pass. It ends abruptly and spontaneously as it began, and at once the patient feels himself again, or merely tired by his experience. There is seldom more discomfort than this during short attacks, the chief trouble being their recurrence at unexpected times, but if the tachycardia persists for several hours, still more if it persists for days, the symptoms of heart failure supervene. Respiratory distress becomes evident, exhaustion is manifest, and pain over the left breast with pain and tenderness from an enlarging liver add to his serious symptoms. Dizziness, nausea, and vomiting are not uncommon accompaniments.

The characteristic sign during an attack is the extreme tachycardia at some fixed rate between 120 and 220 a minute, with a regular and small-volume pulse. The particular rate does not alter with change of posture or with exertion. The further signs which arise in a prolonged attack are simply those of steadily increasing heart failure. Dyspnoea increases even to orthopnoea, and cyanosis appears. The heart becomes dilated and crepitations multiply at the bases of the lungs. The liver swells and

PAROXYSMAL TACHYCARDIA

the legs become oedematous, while the urinary secretion falls. However extreme the symptoms and signs, they rapidly abate with the abrupt cessation of the tachycardia and resumption of the normal rhythm.

Diagnosis. (1) **Between attacks.**—When an attack is not witnessed, close attention to the patient's story often renders a diagnosis possible. He will describe compact attacks in so characteristic a fashion as to exclude the diagnosis of simple palpitation.

Ordinary *palpitation* is a far more common complaint, and often means no more than undue perception of the heart's action. True, it may have some foundation in the occurrence of premature contractions, or in an excessive

separated by means of the electrocardiograph. In proportion to the duration of the attack and the ventricular frequency, the symptoms and signs of heart failure are present. It is a typical attack of heart failure, of sudden onset, depending entirely on extreme tachycardia. The respiratory symptoms and signs have led to a diagnosis of *pneumonia*, the rapid onset of abdominal (liver) enlargement to that of an "*acute abdomen*." Paroxysmal tachycardia is the correct diagnosis in many cases of alleged heart-strain or acute heart failure otherwise unexplained.

A polygraphic tracing will record the rapid regular pulse and permit the counting of its invariable rate (Fig. 68). Pulsus alternans may

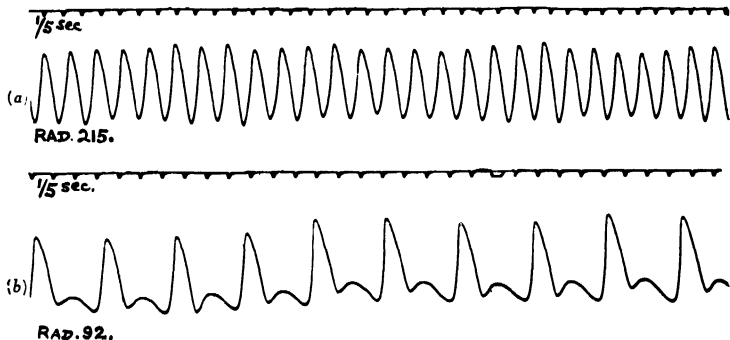


Fig. 68.—Paroxysmal tachycardia. Radial tracings taken (a) during a paroxysm at a rate of 215 beats a minute; and (b) five minutes later when the attack had stopped—the rate is 92 a minute.

reaction of heart-rate to sudden emotion or exertion; yet the rate and rhythm of the heart may be faultless during palpitation and the patient hypersensitive enough to be worried, even by the normal action of the heart. In paroxysmal tachycardia the attack begins with a bound and without any adequate cause, perhaps even waking the patient from his sleep. It lasts for a defined period during which the rapid beating is continuously felt, then it stops suddenly and spontaneously—not gradually as does simple palpitation. Between the paroxysms the patient is well.

(2) **During attacks.**—The high frequency of the pulse attracts attention at once; it is regular, small, and often uncountable. Similar attacks, but with a completely irregular pulse, are paroxysms of *auricular fibrillation*. The variety due to *auricular flutter* can only be

be seen. The jugular curve is atypical, and an electrocardiogram will show an abnormal rhythm arising elsewhere than in the normal pacemaker of the heart (Fig. 69). It may arise in the auricle (auricular tachycardia), in the auriculo-ventricular node (*A-V tachycardia*), and very rarely in the ventricle (ventricular tachycardia).

Prognosis.—In a patient showing no symptoms or signs of heart disease between the attacks, the prognosis is favourable, for in such a case it is very exceptional for an attack to persist long enough to endanger life. Still, it is impossible to forecast the frequency or duration of attacks, unless perhaps from the patient's previous experience, and they certainly tend to recur, though not necessarily with increasing frequency. They may be little more than an inconvenience during many years of active life.

In a patient already a sufferer from chronic heart disease the prognosis is less favourable. The tachycardia tries a damaged heart so severely that if it persists or recurs frequently, heart failure is soon induced, with enlargement of the heart, engorgement of the liver, and increasing cedema. A paroxysm may thus prove to be the terminal event in a case of myocardial or valvular disease.

The prognosis in paroxysmal fibrillation and flutter is discussed with those diseases, for they often merge into the permanent condition.

Treatment.—There is no treatment known to influence the frequency or severity of the paroxysms, except that a course of bromide (10–20 gr., t.d.s.) occasionally seems to reduce their frequency, and the wearing of a firm

PATHOLOGY, CHEMICAL, MODERN DEVELOPMENTS OF.—The modern developments of chemical pathology have been rendered possible only by the advances made in the science of pure chemistry during the last quarter of a century. To the student of medicine the **chemistry of the carbon compounds** is of most interest, and among these the proteins and the carbohydrates rank first. Although we are yet far from synthesizing a typical **protein**, sufficient is known of the composition of proteins to make plain many problems previously difficult to explain. It has now been established that all of them are combinations of amino-acids, the number and varieties of which differ, however, in different proteins. The precipitin test and the pheno-

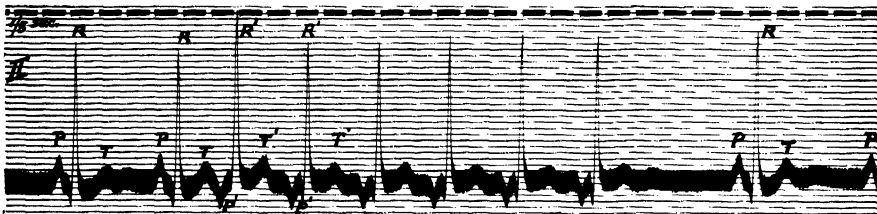


Fig. 69.—Paroxysmal tachycardia. Electrocardiogram showing the whole of a brief paroxysm. The first and second cardiac cycles are normal: P = auricular systole, R = the beginning and T = the end of ventricular systole. Then an abnormal (inverted) P' occurs prematurely, followed by five more, each succeeded by R' T'. These six cycles constitute the paroxysm (rate 120), after which there is a pause, the normal rhythm (rate 75 a minute) then being resumed.

abdominal belt has done so in a few cases. During an attack the patient should rest, and if it persists unduly he should remain in bed until it passes. Pressure on the vagus in the neck, bending down and holding the breath, the induction of vomiting, and other procedures have succeeded in stopping the attack in a few instances, but as a rule one can only await its spontaneous offset. If the circulation becomes embarrassed in a persistent attack, the general treatment is that of heart failure, and venesection, oxygen, morphine may all be useful in relieving symptoms. Digitalis proves to have little or no effect in heart failure due to this condition.

JOHN PARKINSON.

PARRY - ROMBERG DISEASE (see FACIAL HEMIATROPHY).

PATHOLOGY AND BACTERIOLOGY, CLINICAL (see BACTERIOLOGY AND PATHOLOGY, CLINICAL).

mena of anaphylaxis and cytolysis show that even in proteins of the same composition the relationship of the amino-acids may differ, the materials being the same but the design being different. During digestion the proteins of the food are broken down into their constituent amino-acids, some 18 or 20 in all. These are absorbed and carried to the tissues, each of which selects the varieties that it needs, building them into its own structure. The unused balance is carried to the liver, where the amino-acids are split up, the non-nitrogenous part being used for the production of energy and heat, while the nitrogenous part is excreted as urea. If the functions of the liver are being imperfectly performed, unsplit amino-acids may appear in the urine. For normal growth and repair certain amino-acids are essential, and it is for this reason that some substances in which they are lacking cannot be used as the sole protein of the diet. Thus the gliadin of wheat is deficient in lysin; zein, the most

conspicuous protein of maize, does not yield lysin, tryptophan, or glycoecoll; and gelatin contains no tyrosin, tryptophan, or cystin.

Our knowledge of the **carbohydrates** is now fairly complete, and many sugars have been synthesized. Carbohydrates constitute from 60 to 75 per cent. of an average diet, and are the main sources of heat and energy for the body. Deposited as glycogen, they form a proximate store of energy. A small proportion also enters into combination with the proteins and lipoids of the tissues, partly as hexoses and partly as pentoses. The **lipoids** are compounds of fatty acids, the commonest being esters of glycerol. In the body the fats, which form the bulk of the lipoids, act as a further source of heat and energy, the unused excess being deposited in the form of fat as an ultimate store of energy. The presence of a certain amount of carbohydrate is necessary for the normal metabolism of fat. When the liver is depleted of its glycogen the regular degradation of fatty acids is interfered with, acetone bodies appearing in the urine and the symptoms of acidosis developing. It has also been shown that a carbohydrate diet protects animals more efficiently from the action of poisons tending to produce necrosis of the liver than a diet of fats and proteins.

It is now evident that **inorganic salts** play a much more important part in the life processes of the body than had been supposed. The salts which the tissues contain are not merely dissolved in the water of the cells but are combined in part with the organic constituents. It seems probable for instance that proteins take part in vital chemical processes only as ion compounds with inorganic elements. The inorganic salts also regulate the chemical mechanism of the body by the influence they exert on the processes of diffusion and osmosis.

What are termed "**deficiency diseases**" arise from a deficiency, or absence, of one or other of the elements of a normal mixed diet. Beriberi, scurvy, infantile scurvy, and pellagra are now included under this heading; some of them, at least, appear to arise from a lack of certain essential substances in the food known as "vitamins," which are normally present in minute quantities. Vitamins also appear to be necessary for normal growth to take place.

Reactions occur within the organism between substances that do not affect each other outside the body, and proceed in directions which cannot be imitated by ordinary methods. Such activities are believed to be due to the influ-

ence of **ferments** or **enzymes**. No ferment has yet been isolated in a pure state; they are recognized solely by their actions. At one time supposed to be unique, it is now considered that their effect is similar to that of all catalytic agents—that is, they increase the speed of reactions which would go on without them, but at a very much slower rate. Enzyme effects are no exception to the rule that chemical reactions are reversible. There is, therefore, a continuous attempt to establish a state of equilibrium, when the action of the ferment would cease, but, owing to the removal of some of the products of the reaction or the introduction of fresh material, the balance is upset and the process proceeds. Changes of this description are the foundation of normal metabolism, both in the individual cells and in the organism as a whole. Ferments play an important part in many pathological processes such as autolysis, fatty degeneration and fat necrosis, whilst the absence of certain specific ferments is no doubt responsible for the "inborn errors of metabolism," including alkaptonuria, cystinuria, pentosuria, albinism, and probably for certain acquired diseases such as diabetes and gout. The co-ordination and regulation of the metabolism of the body seems to be effected largely by means of enzymes secreted by the glandular structures, under the control of the sympathetic nervous system, and although much of the theory of this aspect of physiology is supported by only slender evidence, there can be no doubt that it has profoundly modified our views of the pathology of a number of diseases, including cretinism, myxœdema, tetany, infantilism, obesity, and glycosuria.

Toxins appear to be closely related to enzymes, and the mechanisms by which anti-toxins, agglutinins, precipitins, opsonins, etc., are produced are similar to those by which the cells protect themselves from the ferments they contain or that originate in other tissues. According to Ehrlich's theory, the action of toxins is purely chemical, the toxin uniting with a cell because some chemical group in its molecule has an affinity for some particular group in the cell protoplasm. An antitoxin consists of protoplasmic groups, capable of combining with the toxin, which have been produced in excess and shed into the circulation. There they combine harmlessly with the toxin, for this can only cause injury to the organism when it unites directly with the protoplasm of structures of prime importance to the life of

the organism. With few exceptions the excreted enzymes exist in the cells where they are produced, in an inactive form which is activated by some specific agent after they have been discharged. Parallel to this is the activation of a normal constituent of the blood, known as "complement," by specific immune bodies formed by the organism in response to the presence of foreign proteins, or "antigens," which are then attacked by the complement. The immune bodies are protoplasmic cell-groups, produced in response to the presence of an antigen in the same way that an antitoxin results from the introduction of a toxin. They unite with the antigen as an antitoxin unites with a toxin, but they differ from antitoxins in having a second receptor by which they can combine with the complement and bring it into relation with the antigen. They are therefore also known as "amboceptors." Complement, like toxins and enzymes, possesses at least two receptors—one, the haptophore, which unites with the amboceptor, and the other, the toxophore, which attacks the antigen.

Bacteriolysis and hæmolytic by serum are explained by this mechanism, and it is also the foundation of the Wassermann complement-fixation reaction. In the former the bacteria, or erythrocytes, are the antigen which gives rise to the formation of specific immune bodies. Sensitized by union with these, the antigen is given a chemical affinity for complement, the toxophore group of which then attacks the bacteria, or the red cells, destroying the one and setting free the hæmoglobin of the other. In the Wassermann reaction for syphilis, the presence in the blood of the specific amboceptors for the virus is shown by the failure of a mixture of the suspected serum with complement and a syphilitic antigen to hæmolyse erythrocytes in the presence of specific hæmolytic amboceptors. Certain pathological conditions of which hæmolytic is a feature are also due to actions of this type. Thus paroxysmal hæmoglobinuria appears to depend upon the presence in the serum of a hæmolytic amboceptor which combines with the red corpuscles of the same individual and sensitizes them for his own complement. In other conditions the mechanism is different. Many bacteria produce hæmolytic substances akin to toxins, which are excreted into the medium in which they grow and directly attack the red cells, thus explaining the anæmia of some infectious diseases. In some forms of poisoning, too, the blood changes with which they are

associated seem to be produced by a direct chemical action of a simple chemical agent on the erythrocytes. The methods of defence employed against non-protein poisons are simpler than those made use of against toxins of a protein nature. Inorganic poisons are rendered inert by oxidation, reduction, or the splitting off of water. Against organic poisons similar reactions are employed, with also the formation of addition compounds. The substances made use of in forming such compounds are all products of normal metabolism, and no one of them is specific; in fact, several may be used to combine with the same poison.

Auto-intoxication may arise from the formation within the body of abnormal substances with poisonous properties, the production in excess of normal toxic compounds, and failure of the usual detoxicating and excretory processes. The chemical nature of the substances causing auto-intoxications has been determined in very few instances, owing probably to there being more than one concerned in most of them. The most important examples of auto-intoxications are cholæmia and uræmia. In **cholæmia** there is a retention of bile-pigments, bile-salts, and the less conspicuous constituents of bile. Their relative toxicity is not agreed upon, but no doubt some of the symptoms of jaundice result from the action of the bile-salts upon the nervous system and blood. The chemical pathology of **uræmia** is in an even less satisfactory position. It is natural to assume that since the kidneys are the chief channel for the removal of nitrogenous waste products, the retention of these, or their antecedents, is responsible for the condition. Urea is non-toxic, and uric acid, the purin bases, and urinary pigments are only slightly poisonous. We do not know all the steps by which amino-acids are converted into urea, but as ammonia and ammonium carbamate are believed to occur, and these are known to be toxic, uræmia has been ascribed to their accumulation in the blood. It is more probable, however, that many and varying intermediate products of nitrogenous metabolism are responsible for the symptoms. In a similar way the available evidence is against the **toxæmias of pregnancy, eclampsia, and acute yellow atrophy** of the liver being due to specific toxins; each is probably caused by several. Failure of the liver to carry out its functions probably plays an important part in the production of these conditions, as it does in **delayed chloroform poisoning, phosphorus**

poisoning, acid intoxication and allied conditions. **Diabetic coma** is closely related to acid intoxication, and is generally ascribed to a diminished capacity of the blood to carry carbon dioxide owing to its carbonate being combined with organic acids formed in the imperfect metabolism of fats. So long as 70-75 gm., or thereabouts, of carbohydrate are utilized daily, only traces of acetone appear in the urine, but when the daily oxidation of carbohydrate falls below that minimum, acetoacetic acid and beta-oxybutyric acid are excreted, combined partly with mineral bases, but chiefly with ammonia. Normally ammonia contributes only 2 to 5 per cent. of the total nitrogen of the urine, but in cases of acidosis the proportion may reach 10, or even 25 per cent., the proportion contributed by urea being correspondingly reduced. This diversion of nitrogen from urea-formation to the production of ammonia is an attempt on the part of the body to maintain the hydrogen ion content of the blood within a normal limit, which can only vary within a very narrow zone of change without producing striking effects on the mechanism of respiration. Physico-chemical studies of the blood have shown that its hydroxyl ion concentration undergoes little change even under conditions approaching extreme acidosis, although its titratable alkalinity may be much reduced.

Alimentary intoxications.—Intoxications arising from the absorption from the alimentary tract of substances formed from its contents by chemical change are commonly referred to as auto-intoxications, but the strict propriety of such a classification is doubtful. Many of the constituents of the digestive juices are toxic, and some of the products of digestion, notably the earlier cleavage products of the proteins, are also poisonous, but there is no evidence that they are responsible for intestinal auto-intoxication. It is, therefore, to the products of bacterial putrefaction and fermentation that we must look for an explanation. Most of the products of intestinal decomposition so far isolated are only mildly toxic, and can probably only produce serious effects as a result of long-continued absorption. Among such are indol, skatol, and phenol, derived from the aromatic radicals of the proteins, and possibly the diamines, putrescine, cadaverine, etc., originating from the fatty acid radicals. Tyramine, formed by the putrefaction of tyrosin, is known to have a marked effect in raising the blood-pressure, and its formation

from animal proteins is regarded by some as the cause of hypertension in man. **Histamine**, formed in a similar way from histidin, has been claimed as the cause of bronchial asthma and urticaria. A hydroxylamine derivative capable of reducing oxyhæmoglobin is said to have been found in the urine in cases of sulphæmoglobinæmia, and enterogenous cyanosis with methæmoglobinæmia is stated to be due to the effect of nitrites formed in the intestine. The sulphur-containing radicals of the proteins give rise to hydrogen sulphide and mercaptans to which some observers have referred pathological conditions. The gases arising from the fermentation of carbohydrates produce discomfort and mechanical difficulties, but have no toxic effect, while the fatty acids and oxyacids originating in the same way are normally oxidized by the tissues. There is evidence, however, that oxaluria may arise from the fermentation of an excess of carbohydrate in the upper intestine. The fatty acids formed in the decomposition of fats irritate the intestine and may reduce the bases in the blood. It has been shown that choline, muscarine, and neurine, which are exceedingly poisonous, may arise in the intestine from the decomposition of lecithin-rich foods and explain the toxic effects produced in some persons by the ingestion of such articles of diet as eggs and brains.

Among **disorders of metabolism**, gout and diabetes take first place, partly on account of their clinical importance and partly owing to the interest and complexity of the phenomena they present. The chemical pathology of **gout**, and the intimately related chemistry of uric acid, have been the subject of many investigations during the past twenty years. It has now been shown that uric acid is one of a group of substances built around a common nucleus. Other substances containing this "purin nucleus" are adenine, guanine, xanthine, and hypo-xanthine. These, the purin bases, are derived chiefly from the nucleoproteins, which may be looked upon as salts of a protein with nucleic acid, the nucleic acid being a combination of purin bases, pyrimidine bases, and carbohydrate radicals. Uric acid does not exist in the nucleo-protein molecule as such, but is readily formed from other purin bases by the action of specific enzymes. In most mammals only a small proportion of the purin bases taken in the food ("exogenous") or set free in the tissues ("endogenous") appears in the urine as uric acid, most being

PATHOLOGY, CHEMICAL, MODERN DEVELOPMENTS OF

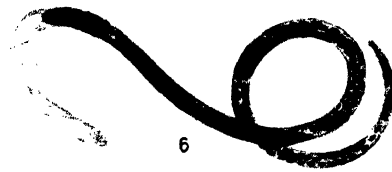
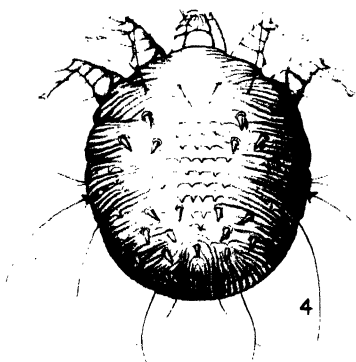
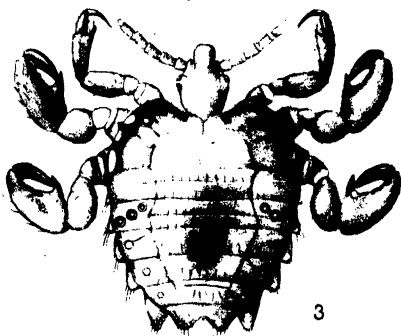
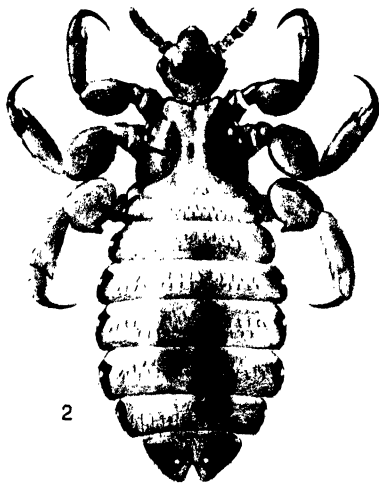
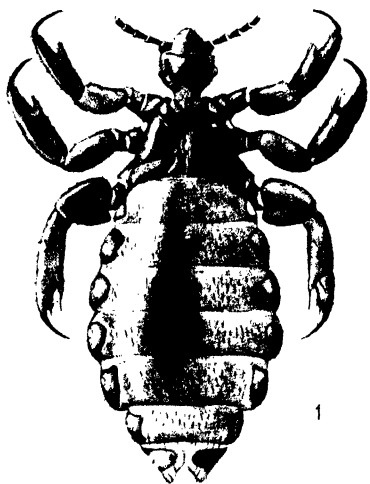
converted into allantoin. Man is an exception to this rule, a considerable proportion of the purin bases being excreted as uric acid. This is in harmony with the observation that the tissues of man have no power to destroy uric acid *in vitro*. It would seem probable, therefore, that the fundamental reason for the existence of gout in man lies in his natural inability to destroy uric acid. Irregular periods of nitrogen retention and loss are also quite constant features of gout, and suggest that it is not uric-acid metabolism alone that is defective. Moreover, higher concentrations of uric acid in the blood may be met with in other conditions without any gouty manifestations. It is, consequently, not unlikely that the precursors of uric acid—the purin bases—are more harmful than uric acid itself and that to them some of the pathological changes and symptoms of gout may be due.

More work has probably been done on the chemical pathology of **diabetes** than upon any other subject, and it is now clear that its discussion involves a consideration of the fate and utilization not only of ingested carbohydrates, but also of the fats and proteins. In the early stages of most cases of diabetes it is true that there appears to be merely a lowered tolerance of carbohydrates, first of sugar and then of starches, but if this be not controlled, the metabolism of proteins and fats eventually becomes obviously defective, the disturbance spreading until the metabolic processes concerned in all the proximate principles of the food are involved. When the diabetic organism is unable to assimilate carbohydrates it is deprived of its most important source of energy, and to make good that deficiency it is bound to rely more and more on sugar of protein origin. At first the circulating amino-acids are broken down and used for this purpose, but later sugar derived from the proteins of the tissues is called into use. Meanwhile the amino-acid sugar of the food, and finally that derived from the tissues, is passed in the urine unused. At the same time the fats are more and more incompletely oxidized, and increasing quantities of lower fatty acids and acetone are excreted. True diabetes cannot, therefore, be regarded simply as a failure of carbohydrate metabolism; the secondary disturbances of protein and fat metabolism which follow in its train must be taken into account also, and be considered in arranging the treatment and giving a prognosis.

The application of chemical methods to

the diagnosis of disease has made rapid strides in recent years. The excreta, the blood, puncture fluids, air from the lungs, and the contents of the stomach and upper intestine are now submitted to chemical examination for this purpose. The methods employed in investigating the urine and feces are described elsewhere (see URINE, EXAMINATION OF, and FÆCES, EXAMINATION OF), but it may be pointed out that it is largely owing to the substitution of quantitative for qualitative analysis that progress has been made. The introduction recently of comparatively simple methods by which sugar, uric acid, urea, the acetone bodies, cholesterol, etc., can be estimated in small quantities of blood has opened up a field for investigations which seem likely to throw new light on the pathology of gout, diabetes, and other metabolic disorders, beside aiding in their diagnosis and treatment. The more complicated methods of Abderhalden for revealing protective ferments in the serum have widened still further the scope of chemical investigation in this direction.

Chemical methods in dietetics.—Our knowledge of the metabolic processes, the composition of food materials, and the way in which they are digested and absorbed, has reached a stage when it is possible to determine with considerable accuracy the dietetic needs of the body under various conditions and to adjust the food supply to meet particular digestive or metabolic difficulties. **Scientific regulation of the diet** finds its chief value in the treatment of disorders of metabolism, but to carry it out successfully it must be done cautiously and with a due sense of responsibility. It is a serious matter to restrict materially the intake of any class of food, and when doing so we must remember that, although we may be eliminating one evil, we may possibly be initiating, or accentuating, another, by diverting metabolism along channels which are unable to meet the strain. There can be no doubt, for instance, that a restriction of the carbohydrate intake is of value in diabetes, but to give a patient merely a list of starchy food he is to avoid and another of fats and proteins he may take is often to court disaster. Proteins and fat will then form the major part of the diet in most instances, with the result that disturbances of metabolism which may shorten life more speedily than a moderate glycosuria are likely to supervene. To avoid this the diet should be worked out and prescribed on a quantitative as well as a qualitative basis.



1, *Pediculus capitis*, female, $\times 25$.

3, *Pediculus* (*Phthirus*) *pubis*, female, $\times 45$.

5, *Microfilaria loa*, $\times 500$.

2, *Pediculus corporis*, female, $\times 35$.

4, *Sarcoptes scabiei*, female, $\times 125$.

6, *Microfilaria bancrofti*, $\times 500$.

PEDICULOSIS

tive basis suited to the tolerance of the patient. Under abnormal conditions it is just as important to know the proper dosage of food as the correct dosage of drugs, for custom and appetite cannot be relied upon as safe guides. Regulation of the food supply by its caloric value is more difficult in children than in adults, and this is one of the reasons why the dietetic treatment of metabolic disorders is often less satisfactory in children than in persons who have reached full growth.

Modern chemistry in relation to therapeutics.—Beside providing a rational basis for the use of many drugs which had been used empirically with success, chemical pathology has extended the field of therapeutics in fresh directions, whilst restricting it in others, by showing that some morbid symptoms are merely manifestations of protective mechanisms which need to be encouraged rather than suppressed. From an understanding of Nature's methods in combating disease we are now able to copy them and assist her efforts by timely interference. The vaccine-therapy and preventive inoculation of infectious disorders furnish striking examples, but the brilliant results obtained with salvarsan, atoxyl and other synthetically prepared drugs indicate that the application of strictly chemical methods to therapeutics is likely to place even more potent weapons at our disposal in the fight against disease in the future.

P. J. CAMMIDGE.

PATHOPHOBIA (see PSYCHASTHENIA).

PEDICULOSIS.—There are three species of pediculi which attack the human body—*Pediculus capitis*, *P. corporis* or *vestimenti*, and *P. (Phthirus) pubis*. Each of these parasites, with some exceptions on the part of *P. pubis* and possibly of *P. corporis*, limits its field of operations to its own particular part of the body, rarely straying to other parts. The presence of these parasites, and the itching, scratching, and secondary lesions to which they give rise, constitute the disease known as pediculosis.

1. **Pediculosis of the head.**—The head-louse (PLATE 24, Fig. 1) measures about 2 mm. in length and 0.7 mm. in breadth, the female being larger than the male. It has an elliptical body of greyish-brown colour, with six legs attached to the thoracic portion and a triangular-shaped head with two antennæ. The female deposits her ova on a hair close to the root, the pear-shaped egg or nit being attached to the hair-shaft by a glutinous collar and

having an operculum or lid at its free extremity through which the young emerge in about a week. Pediculosis of the scalp gives rise to itching, and is commonly complicated by the presence of impetiginous crusts on the scalp and face and enlargement of the occipital or cervical glands. Occasionally there is an erythematopapular eruption on the back of the neck and shoulders or on the trunk.

Diagnosis.—Pediculi may be seen, or, if these are absent, nits will be found adhering closely to the hairs. If impetiginous lesions are present the hair is matted together with thick brown crusts and there will probably be yellow scabs on the face or elsewhere. The scales of *seborrhœa* or *psoriasis* may be mistaken for nits but are easily distinguished by the fact that they are not attached to the hair and cannot be drawn along it. *Syphilitic crusts* show underlying ulceration, and *ring-worm* and *favus* are distinguished by short broken hairs and yellow cups respectively.

Treatment.—The hair should be soaked in oil of sassafras or a 1:40 solution of carbolic acid at night and washed in the morning with soft soap or equal parts of soft soapsuds and paraffin. This treatment will kill the pediculi and remove the crusts. Ammoniated mercury ointment may then be applied. Nits can be removed by a fine-tooth comb after soaking them with vinegar or weak acetic acid, or, if few in number, by cutting away the hairs to which they are attached.

2. **Pediculosis of the body.**—The body-louse (PLATE 24, Fig. 2) is similar to but larger than the head-louse, the female being about 3.3 mm. in length and 1.4 mm. in breadth. It is a dirty-grey colour varying in tint according to that of its host; when engorged with blood it is brownish. It lives in the folds and seams of the underclothing, which it grasps with its legs while feeding on the skin. The ova hatch out in about a week, but temperature plays an important part in their development and they are capable of hatching out up to a period of at least 35–40 days after they are laid (Warburton, quoted by Shipley). Body-lice affect chiefly the poorer classes when there is neglect of personal cleanliness; they are also a common pest in armies and camps and under conditions which render proper ablutions and change of underclothing impossible.

Symptomatology and diagnosis.—The symptoms produced are severe itching and scratching of the skin, resulting in the formation

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of blood-crusts, wheals, pus-infections, etc. Small hæmorrhagic spots which do not disappear on pressure are often present and correspond to the mouths of the sweat-glands through which the pediculus has drawn blood. In cases of long standing the skin may become leathery and deeply pigmented, a state of affairs which has given rise to the name "vagabond's disease."

The diagnosis is made by the finding of pediculi in the seams of the underclothing or, if these are absent, by the presence of hæmorrhagic points, scratch-marks, blood-crusts, pus-infections and other evidence of scratching on the upper part of the back and shoulders or in other parts of the body within reach of the finger-nails. The character and distribution of these lesions serve to distinguish them from those of *scabies* or *urticaria*, and, when pigmentation is present, from *Addison's disease*, *chronic arsenical poisoning*, or other causes of pigmentation.

Treatment.—This consists in thorough disinfection of the underclothing by steaming, baking, or ironing, and the inunction of an antiparasitic ointment such as ung. staphisagrie or ung. hydrarg. ammoniata, after a hot bath. An old-fashioned remedy, as a means both of prevention and cure, is powdered sulphur dusted over the underclothing and bedding or worn in a small muslin bag next the skin. A more efficacious powder is one used in the Army, consisting of naphthalin, creosote, and iodoform, and known as N.C.I. powder. The extermination of body-lice acquires an importance other than merely dermatological from the fact that they are carriers of relapsing and typhus fever and of trench fever.

3. Pediculosis of the pubic region.—*P. pubis*, the crab-louse (PLATE 24, Fig. 3), is smaller and shorter than the head- or the body-louse, measuring about 1-2 mm. either way. It possesses six jointed legs terminating in claws with which it tightly grasps the shaft of the hair. In colour it is grey, but becomes yellow or brownish when engorged with blood. The female, much larger than the male, deposits eggs to the number of 15-20, which take about a week to hatch out. The nits are arranged in rows along the shafts of the hairs, and are similar in shape but smaller and rather darker in colour than those of *P. capitis*. In exceptional cases the crab-louse is found on the hairs of the thighs, chest, axillæ, and perineum, and sometimes on

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the eyelashes. The disease is conveyed from one person to another by close contact, and in the majority of cases by means of sexual intercourse, but there are also external sources of infection such as unclean linen, lavatory seats, etc.

Symptomatology.—There may be little itching, or this may be severe, leading to scratching and excoriation of the skin of the affected part. Small cedematous papules and pustules appear, and, especially when strong antiparasitic preparations are used, an acute eczematous dermatitis may be set up and spread beyond the area affected by the pediculi. The pediculus also causes in some people small bluish stains known as "maculæ carulæ" or "taches bleuâtres," due to a pigment excreted by the parasite, which were at one time thought to be peculiar to typhoid fever.

Diagnosis.—A papular or eczematous dermatitis limited to the pubic region is always suggestive of the presence of the pediculus, and on close inspection the parasites or their ova will be seen at the roots of the hairs. Occasionally a *follicular impetigo* or a *localized eczema* may occur in this part of the body independently of pediculosis, and an acute *mercurial dermatitis* may mask the real cause of the disease.

Treatment.—The usual application, viz. blue mercurial ointment, is unnecessarily severe. Unguentum hydrarg. ammoniat. (5 per cent.), applied after washing with soft soap, is sufficiently strong to kill the pediculi without setting up a dermatitis. Other effective applications are weak perchloride of mercury or carbolic lotion, paraffin, xylol, and petrol. Shaving is a rapid method of cure, but is inadvisable owing to subsequent irritation set up by the newly-growing hair. In pediculosis of the eyelashes the parasites and ova can be removed with forceps.

S. E. DORE.

PELIOSIS RHEUMATICA (see PURPURA).

PELLAGRA (*syn.* Mal de la Rosa, Alpine Scurvy, Asturian Rose, Psilosis Pigmentosa).—A chronic endemic disease of obscure etiology, running a prolonged course, and associated, during the spring and autumn months, with symptoms referred to the skin, alimentary canal and central nervous system. These symptoms recur yearly during the same season, but are in abeyance during the winter months. As far as is known, the disease is not com-

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municable from man to man, and affects mostly agricultural labourers of a poor class.

Geographical distribution.—Pellagra appears to be widely distributed in Southern Europe and North Africa. In recent years it has been recognized as occurring commonly in the United States.

Europe.—In Spain pellagra has been known since the time of Casal in 1735, and it has been reported from Northern Portugal. At the beginning of the last century, it was apparently recognized in the South of France. It has been the scourge of Italy since 1750, when it was first detected; at the present time it is estimated that there are over 100,000 pellagrins in that country. It is known to occur in parts of Austria and Hungary, Russia, Dalmatia, Bulgaria, Greece, and Rumania, and a few indigenous cases have also been reported in the British Isles.

Asia.—Our knowledge of the distribution of pellagra in this continent is by no means complete. It was extremely prevalent among the Turkish prisoners from Asia Minor, Syria, Palestine, and Arabia during the Great War, and even occurred among the Germans captured in that theatre; scattered cases have been recorded from India, Singapore, and the Philippines.

Africa.—Pellagra is prevalent in Egypt, especially in the Delta, where it accounts for a great amount of insanity (Warnock); it is found in Tunis and Algiers, the Red Sea Provinces, Nyasaland, and Zululand, as well as in Cape Colony. Sandwith has recorded that some of the negroes interned on Robben Island are pellagrins.

America.—The disease appears to be common among the negroes and "mean whites" in the West Indies, Southern United States, Mexico, and Central America, where it was first noted in 1907, and where in 1916 the number of cases was estimated at 150,000.

Australia.—Pellagra has been reported from New Caledonia.

Etiology.—Apparently in the countries in which pellagra is endemic, people of all ages and both sexes are liable, especially agricultural labourers; this is certainly the case in Italy, and from the records of the Southern United States the same rule would seem to apply. The affluent are seldom affected.

Two salient facts emerge: (1) that the acute symptoms manifest a striking seasonal variation, a spring incidence occurring during February and March in North Africa, and

during May and June in Europe, and an autumn one in September and October; (2) that the disease as a whole exhibits waves of activity followed by periods of quiescence.

Notwithstanding the immense amount of work which has been done on this disease during recent years, especially in the United States, and in spite of the numerous theories which have been advanced, all of which possess their adherents, we have at present no certain knowledge of the true cause of pellagra. The leading theories are these:—

1. **The intoxication theory.**—The connexion between the incidence of pellagra in a community and the consumption of diseased maize was first put forward by Lombroso and Bellardini in 1871. It has been the basis of public measures taken in Italy and Southern Europe against this disease and has probably the greatest number of adherents at the present day. The grain is supposed to be damaged through damp or through being insufficiently dried, and to be the medium of growth for poisonous fungi,—*Penicillium glaucum*, *Mucor racemosus*, *Aspergillus niger*, and *A. fumigatus*, with the addition of other micro-organisms and yeasts.

Lombroso isolated from alcoholic and watery extracts of damaged grain an alkaloid called *pellagrosein* which produced various poisonous effects in animals, but there are no experiments conclusive of its ability to reproduce, in animals, or even in higher apes, the symptom-complex recognizable clinically as pellagra. The disease does occur commonly in some countries, such as Italy, Lower Egypt, Barbados, and the Southern United States, in which maize bread forms the staple article of diet, but the theory leaves unexplained the sporadic cases of undoubted pellagra which have occurred in the British Isles, Germany, Poland, China, and Nyasaland, where maize is not consumed to the same extent.

2. **The infection theory.**—Various micro-organisms have been suspected of causing a septicæmia and therefore of being responsible for this disease. Ceni in 1902 believed it to be due to a generalized infection with fungi, such as *Aspergillus fumigatus* and *A. flavescens*; but, apart from the fact that these fungi are not present in the tissues at autopsy, it is pointed out that none of the symptoms resemble those of other known mycoses.

Tizzoni described a *Streptobacillus pellagrarum* in the blood and fæces, but recent researches have failed to substantiate his work.

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Since 1905 Sambon has criticized the maize theory and substituted for it, on hypothetical grounds, and mainly because of a supposed clinical resemblance between pellagra and trypanosomiasis, a theory that pellagra is a protozoal disease, carried from man to man by the agency of biting flies of the genus *Simulium*. It cannot be said that the hypothesis rests on any sure basis, firstly, because pellagra exists in many places, for instance Barbados, where simuliidæ do not exist at all, or in small numbers, and secondly because during the war in the Near East there were no instances of transmission of the disease to British troops, in spite of their close association for three years with Armenian refugees and Turkish prisoners of war in whom the disease was rife.

Finally, Goldberger's work in 1917 in the United States, in which he failed to communicate the disease to volunteers, after numerous feeding experiments with skin scales and excreta, should dispose of the belief in its transmissibility.

3. The food-deficiency theory.—This theory is gradually gaining ground, and is the only one which rests on any scientific foundation. It has been pointed out that there are certain resemblances between known food-deficiency diseases, such as beriberi and scurvy, which are due to lack of vitamins, and pellagra. For instance, in beriberi certain nerve degenerations comparable to those of pellagra are present. My own personal experience tends to favour this theory. Signs of scurvy, such as spongy gums and hæmorrhages, were observed in some pellagrous Turkish prisoners, and others exhibited an œdema and a peripheral neuritis suggestive of beriberi. A deficiency of certain elements of diet, as Vedder has pointed out, can be demonstrated in most pellagrins; the facts that it occurs commonly in an impoverished population, that it was a disease in Armenia and Turkey during the Great War, and that the same thing seems to have happened in France during the Napoleonic campaigns, would support this view.

The results of the very important experiments of Goldberger and Wheeler in America are in favour of a food-deficiency hypothesis. These investigators conducted two series of experiments—one in which they produced pellagrous symptoms in six out of eleven convicts who volunteered to live on a protein-free diet for six months; the other in which, by substituting a rich protein for an almost

farinaceous diet, they banished pellagra from two orphanages within a year.

The investigations of Wilson and Roaf in Egypt on metabolism in pellagra would seem to indicate that there is a deficient nitrogen excretion in pellagrins dependent, it would appear, on the amount of available biological proteins assimilated. They also show that in a susceptible subject, or in one whose process of metabolism has been vitiated by such alimentary diseases as dysentery, or whose protein needs have been increased by physical exhaustion, if once the balance of protein metabolism has been disturbed through a food deficiency, the symptoms of pellagra occur, even should the patient be subsequently fed on a suitable diet. There is a connexion between this hypothesis and the maize theory, for the disease is commonest in those whose cereal diet, whether of maize or of millet, is either deficient in protein or is lacking in the hexone-bases "tryptophane" and "lysine," substances which are present in all proteins of true biological value.

Finally, recent work would suggest some connexion between the symptoms of pellagra and suprarenal secretion. As pointed out by Rondoni and McCarrison, the suprarenals do not waste as a result of protein starvation, and at autopsy I have found hæmorrhages into the medulla and various degenerations of the cortex of the gland. These observations, as well as the resemblance of pellagrous pigmentation to that of Addison's disease, indicate that further investigations on these lines are desirable.

One important fact has to be recorded against the food-deficiency theory. In November, 1918, a certain number of German prisoners of war from Palestine developed acute symptoms of pellagra. As far as could be ascertained, they had in no way been starved during the preceding three years; according to their own statements they had always been provided with ample fresh meat, eggs, and beer (Enright).

Pathology.—Apart from a general atrophy of all organs, there is little to be seen after death. Patchy degenerations are found in the brain cortex, basal ganglia and spinal cord, without any special distribution.

Symptomatology.—The course of an average case of pellagra is generally one of years rather than of months. As noted originally by Strambio, the disease does not run a regular course, except in so far that the acute exacerbations reappear every spring and autumn. Initial symptoms are a general feeling of



**PLATE 25.—A CASE OF PELLAGRA, WITH “BUTTERFLY”
PATCH ON NOSE AND MALAR REGION.**

(Dr. A. D. Bigland's case.)

debility, spinal pains, dyspepsia and dysphagia. These phenomena may recur for years without the appearance of skin manifestations.

Cutaneous symptoms.—These appear especially on the feet, hands, face, and neck. That the rash is actually dependent upon the actinic rays may be seen in the case of Moslem races, who wear the national pointed slippers, for in them it becomes apparent only on that part of the foot uncovered by the shoe. It is V-shaped on the neck and chest in those who work with shirt unbuttoned. On the face it appears as a butterfly patch on the cheeks and bridge of the nose in much the same way as lupus erythematosus (Plate 25); on each side of the nose there may be a characteristic prominence of the sebaceous glands. On the neck the rash resembles a collar or rosary. There are patches of roughened pigmented skin on the elbows, the backs of the hands, the dorsa of the feet and on the shins. There is at first a diffuse, symmetrical erythema with oedema, swelling of the fingers or toes, and perhaps bullæ. The itching and burning of the affected parts may be intense. On the subsidence of the acute stage the skin assumes a brownish-red colour, and a state of keratosis supervenes with scaly desquamation, leaving behind a white atrophic patch. In the hands the wrinkling so produced makes them resemble the hands of washerwomen or of an old man. The nails become atrophied and cracked.

Gastro-intestinal symptoms.—Acute gastric and intestinal symptoms generally coincide with exacerbations of the skin rash. Pyrosis, gastralgia, achlorhydria, and anorexia are accompanied by increased peristalsis and diarrhoea with flatus and large frothy stools. The mouth becomes inflamed and the tongue red and fissured, especially at the tip and sides; indeed the terminal symptoms may resemble those of sprue. The urine is of low specific gravity, deficient in urea, and generally contains indican. Acetonuria may be present.

Nervous symptoms.—The motor nerves are affected, as is shown by various muscular weaknesses and pareses. Spastic paraplegia with ataxia and Romberg's sign may be met with. Epileptiform convulsions are rare. There may be tingling and burning in various areas, which may alternate with spots of anæsthesia. The reflexes are exaggerated, especially the knee-jerk. Diplopia and photophobia occur, and are associated with mental disturbance, especially with profound melancholia and suicidal tendencies. Other cases show periods of exalta-

tion not unlike those of general paralysis. Most chronic pellagrins become completely demented.

Terminal symptoms.—Finally, the patient becomes anæmic, wasted, bedridden, and almost completely paralysed, with incontinence of urine and feces; his end is generally hastened by an intercurrent disease, such as bacillary dysentery.

An acute form occurs and is known as pellagra-typhus. The symptoms resemble those of typhoid fever, with low delirium and pyrexia (100° – 104° F.), which terminate in fourteen days.

Diagnosis.—Pellagra may be mistaken for a multitude of diseases by the uninitiated. The skin rash must be distinguished from acrodermia, erythema solare, eczema, trade dermatitis, and syphilis. The gastro-intestinal symptoms may be confused with those of sprue, in which disease, however, there are no skin manifestations. The nervous symptoms may suggest hysteria, general paralysis, ergotism, or lathyrism.

Prognosis.—The prognosis is always serious. The death-rate from the initial attack has been estimated as 15.8 per cent. by Siler and Garrison. The duration of the disease is protracted, but very obstinate cases may recover, though relapses are common.

Treatment.—There is unfortunately little to be said about treatment. No specific exists. There is some evidence that if the disease is treated early by giving the patient a liberal proteid diet, the symptoms will not recur. Arsenic, iron, and strychnine have all been lauded, but their exhibition is quite empirical.

P. MANSON-BAHR.

PELVIC CELLULITIS (*syn.* Parametritis).—The pelvic cellular tissue is continuous with the connective tissue lying just external to the peritoneum on its whole surface. In some situations it is thick, in others it is reduced to a fine layer. In the pelvis it is thick and forms a packing between the structures that lie below the peritoneum; it surrounds the vaginal vault and passes outwards in well-marked bands between the layers of the broad ligaments and backwards under the utero-sacral ligaments. It is continuous, when traced forwards over and on either side of the bladder, with the extraperitoneal tissue of the abdominal wall and behind with the retroperitoneal tissue.

The vessels traverse the cellular tissue, and beyond the limits of the pelvis carry out pro-

PELVIC CELLULITIS

longations of it in the form of their sheaths; the ureters also are embedded in cellular tissue continuous with that in the pelvis throughout their whole length.

Etiology.—Inflammation of the cellular tissue of the pelvis may be primary or secondary. *Secondary* pelvic cellulitis occurs in connexion with conditions that cause pelvic peritonitis, such as pyosalpinx and abscess of the ovaries. It is of only secondary importance in these cases and subsides quickly after the primary cause of the inflammation has been removed. *Primary* pelvic cellulitis is caused by direct infection of the exposed cellular tissue, and is probably always due to streptococci. The colon bacillus and the staphylococcus are often found with the streptococcus pyogenes in these cases, and sometimes one or other alone, so it is possible that these organisms may be the sole cause of the condition in some cases.

The great majority of cases of pelvic inflammation are due to pelvic peritonitis, with perhaps some secondary pelvic cellulitis, primary pelvic cellulitis being a much less common affection.

As in cellulitis of the subcutaneous tissues, the organisms find their entrance by some wound in the cellular tissue; this is most likely to be followed by inflammation when the vitality of the tissues has been reduced by bruising.

The pelvic cellular tissue is most commonly infected as a result of labour at or near full term, when lacerations of the cervix, vaginal walls, and lower segment of the uterus are most likely to occur. In some cases the infection may travel through the wall of the uterus into the cellular tissue between the layers of the broad ligament. Any surgical operation, such as hysterectomy or vaginal incision, that opens the cellular tissue may be followed by cellulitis.

The pelvic cellular tissue may become infected from the rectum, and this possibility should not be overlooked in cases of doubtful origin.

Pathology.—When the cellular tissue is infected it becomes engorged with blood, and there is exudation of lymph, causing swelling and oedema. Induration follows, and the part involved may become so hard in some cases as to resemble cartilage to the touch.

The inflammation may resolve, or go on to suppuration. Most frequently it spreads forwards and reaches the abdominal wall above the inner half of Poupart's ligament, where it forms an indurated area which can be felt

extending upwards in the abdominal wall for a variable distance, not uncommonly as much as two or three inches. It may spread backwards along the utero-sacral ligaments and involve the cellular tissue around the rectum, or may spread laterally into the iliac fossa. The tissue surrounding the cervix is frequently involved, but it is uncommon to find the inflammation extending farther beyond the mid-line. Extension may take place along the course of the ureter and reach the kidney region.

Even when the cellular tissue is extensively affected, the inflammation may resolve, leaving no traces behind it. Suppuration, however, may occur, usually a single abscess, but in some cases several independent collections of pus, being formed. The pus gradually makes its way to the surface, but the process is often very slow, and may occupy six or eight weeks or more. The abscess most commonly points above the inner half of Poupart's ligament, but may track backwards or outwards, involving the cellular tissue behind the peritoneum or in the iliac fossa. In these cases it may reach Scarpa's triangle or the buttock by following the femoral or gluteal vessels, or may appear near the anterior superior spine. If it travels along the ureter a perinephritic abscess may form, or the skin may become involved at some lower level above the iliac crest. Occasionally the abscess discharges its contents into the vagina, rectum, or bladder, but these are quite unusual occurrences.

Sometimes an abscess develops in some remote part of the cellular tissue—most commonly in the kidney region—after the inflammation in the pelvis has subsided. In some cases of infection of the cellular tissue the organism is so virulent or the resistance of the tissues so feeble that there is widespread acute inflammation of the cellular tissue without any definite induration or pus-formation, and death may occur rapidly with signs of acute septicæmia.

Symptomatology.—The onset of an attack of pelvic cellulitis is often marked by a rigor within two or three days of infection, but this symptom may be deferred longer. The temperature is always raised and the pulse-rate increased. Pain is not usually noticed, and is probably due, when present, to involvement of the outlying peritoneum. When resolution occurs the symptoms gradually subside, but if suppuration follows, the temperature remains high, the tongue is furred, the appetite goes, and there is marked loss of flesh. The patient

PELVIC CELLULITIS

becomes depressed, pale and sallow, and looks extremely ill. The bowels are usually constipated, but there may be diarrhoea. If the cellular tissue over the psoas and iliacus muscles becomes involved, the thigh on that side is flexed to relax the muscles, and so relieve the discomfort. The existence of pelvic cellulitis is sometimes overlooked until the patient begins to walk, when the stretching of the fascia on these muscles causes pain and calls attention to the condition.

When the tissue around the rectum is involved there is often a clear mucinous discharge from the anus.

Diagnosis.—In the early days of pelvic cellulitis no certainty in diagnosis is possible. There is a history of a recent labour, or of some operation that has opened up the cellular tissue of the pelvis, followed, usually within three days but sometimes after a week or ten days, by a rigor with increased temperature and pulse-rate. A rigor does not occur in all cases, but the temperature is always irregular and the pulse quickened. There is as a rule no pain, and the local signs are at first indefinite. On examination the vaginal vault may be found tender and hot, and marked pulsation of vessels in the fornices may be noticed, but beyond these signs there is nothing to attract attention to the cellular tissue.

After some days the affected tissue becomes swollen and œdematous from the exudation of lymph; the enlargement can then be detected on examination, and later, when induration has set in, this is much more evident to the examining finger.

If the infection has entered through a laceration of the cervix or vaginal wall, the tissue in the base of the broad ligament on the side of the laceration will be the part first involved. The vaginal fornix on that side will be depressed, and attempts to move the cervix will cause pain. Usually there is involvement of the cellular tissue surrounding the cervix, so that induration can be detected at the attachment of the vaginal wall to the cervix. Pressure on the uterus from the abdomen will cause pain in these cases by stretching the inflamed cellular tissue near the cervix.

The inflammation most frequently spreads outwards along the base of the affected broad ligament and then forwards under the peritoneum to the abdominal wall, where an indurated tender mass can be felt above Poupart's ligament. Less commonly the inflammation passes backwards in the tissue

under the utero-sacral ligaments and around the rectum; when this has happened, it is much more easily recognized by rectal than by vaginal examination. The rectum is felt to be surrounded by the indurated tissue, whereas often, per vaginam, only some fullness and tenderness at the back of the pelvis is detected.

When the infection traverses the wall of the uterus itself and cellulitis develops in the broad ligament at a higher level than when the cervix is the seat of infection, a mass can be felt at the side of the uterus and apparently continuous with it. A mass in this position sometimes displaces the uterus to the opposite side; it is not fixed, and can be moved backwards and forwards to some extent with the uterus.

In cases of pelvic cellulitis there is no swelling to be found in Douglas's pouch unless there is associated pelvic peritonitis.

Suppuration, when it occurs, may be suspected from the general condition of the patient, the long-continued fever, and the increase in the size of the inflamed mass. As the abscess approaches the surface, œdema of the skin and an area of softening in the indurated mass indicate the presence and position of the pus; but if the abscess forms deeply in the pelvis it is extremely difficult or may be impossible to ascertain its position unless some soft spot can be reached from the rectum or vagina.

Prognosis.—Except in the fulminating type, which is fortunately rare, the prognosis in pelvic cellulitis is good. In the very acute cases the signs and symptoms are those of severe septicæmia, and the patient usually succumbs within four or five days. Occasionally cases are met with in which there are definite signs of local reaction in the cellular tissue, but which in other respects resemble cases of puerperal septicæmia. These often end fatally after two or three weeks without any perceptible change or increase in the cellulitic mass.

In the great majority of cases in which an inflammatory mass develops in the cellular tissue of the pelvis a favourable termination may be expected. In many, after a week or ten days of illness with a raised temperature, the symptoms gradually subside, and resolution of the mass takes place without leaving any serious effects. Even in those cases in which suppuration occurs it is unusual for any important permanent damage to be left. The amount of damage caused depends on the direction in which the infection travels to

the cellular tissue; if it spreads forwards, as is most common, the pus can reach the surface above Poupart's ligament without extensive burrowing, but if outwards or backwards it has to make its way under the peritoneum over the muscles in the iliac fossa or strip the peritoneum at the back of the pelvis. In these cases the pus takes a long course to reach the surface. The inflammation may involve the psoas and iliacus muscles and give rise to some permanent shortening unless great care is taken in treatment, or the pus may make its way along the sheaths of the vessels, and involvement of muscles in the thigh or buttock may result.

I have known the hip-joint on the affected side to become infected, in one case with bony ankylosis as the result. Where an abscess slowly makes its way backwards under the peritoneum, it is common for it to give rise to local peritonitis, and the adhesions to the bowel so produced may remain and give trouble at any time. As a rule, however, as soon as the pus finds an outlet there is a very rapid improvement in the patient's general condition, the abscess cavity quickly closes, and no bad results remain.

The uterus itself is not damaged, nor are its appendages, unless there has been associated pelvic peritonitis, when adhesions may have closed the fimbriated ends of the tubes.

Puerperal salpingitis seems, in some cases at least, to be the result of a spread of infection to the tube through the cellular tissue of its broad ligament; in such cases the damage to the tube will in all probability be permanent. The scarring in the cellular tissue due to the healing of an abscess may draw the cervix over to the affected side, but this is of no importance.

Treatment.—As pelvic cellulitis is due to infection of damaged cellular tissue, its occurrence should be prevented in the great majority of cases. Thorough aseptic precautions are of course necessary both in the conduct of labour and in any surgical operation. If organisms were not introduced from without, primary pelvic cellulitis would practically disappear, though an occasional case due to auto-infection would occur from time to time. In operating in the pelvis, as well as in managing labour, it is important to avoid bruising and tearing of the cellular tissue by roughness or too hasty delivery. When once infection of the cellular tissue has occurred, very little can be done to modify the course of the disease.

Early in an attack, before the signs have had time to develop, and before a certain diagnosis

can be made, the case must be treated as one of some form of infection following labour or operation.

In puerperal cases a swab should be taken from the cervical canal in order that a vaccine may be prepared for use if necessary. A dose of 20 or 25 c.c. of antistreptococcic serum should be given, and repeated two or three times if the first has a favourable effect on the patient's general condition.

When the signs have developed, the object of treatment is to hasten resolution of the inflammation and to keep up the patient's strength for what may be a long and tedious illness.

A saline aperient should be ordered to ensure a daily action of the bowels, and a liberal diet allowed if the patient can take it.

Hot vaginal irrigation seems to have a beneficial influence in hastening resolution, and douches of about four pints should be given twice a day as hot as the patient can comfortably bear them. If there is pain in the lower abdomen, or if induration can be felt in the abdominal wall, hot fomentations should be applied.

Tampons containing glycerin or glycerin and ichthylol have been advocated, but their value is very doubtful. As pain is not usually serious, drugs for its relief are not needed as a rule, and, when necessary, should be used as sparingly as possible on account of their depressing effect.

When suppuration occurs the patient should be encouraged to take good nourishing food; there is no objection to such solid foods as chicken and fish. Alcohol in moderate quantities is valuable in these cases.

As soon as softening in the indurated mass indicates the approach of pus to the surface or to the vaginal wall, an incision should be made and a large drainage-tube inserted. Before there is definite evidence of the abscess being near the surface it is unwise to attempt to find it by incision. Especially is this so if it appears to be likely that the peritoneal cavity must be opened in exploring the mass, because there is great danger in this event of infecting the peritoneum.

J. P. HEDLEY.

PELVIC ORGANS, FEMALE, DISPLACEMENTS OF.—For many years the treatment of the diseases of women consisted largely in the use of pessaries for the correction of displacements. Then came many advances in the pathology of pelvic disease

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including those made possible by the growth of bacteriology. Further, when gynecologists had shown the possibilities of abdominal surgery, they devoted themselves largely to the development of this branch of their art. Thus displacements have lost pride of place among the diseases of women. But, although they do not endanger life, they cause so much

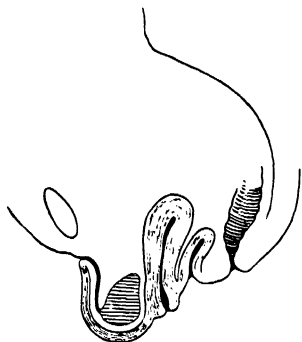


Fig. 70.—Prolapsus uteri: anterior vaginal wall everted from below upwards, followed by cervix.

forward and nearer to the vaginal orifice than usual; it points downwards and forwards, and the body of the uterus is felt behind and not in front of it. In other words, the uterus is retroverted; thus, in early prolapse, cystocele and retroversion are combined. In the *second stage* the anterior vaginal wall is entirely everted from below upwards and the os uteri is at the vaginal outlet. Thus the anterior vaginal fornix is absent. The upper part of the posterior vaginal wall is inverted from above downwards so that the posterior vaginal fornix is only half its usual distance from the margin of the perineum. In the *third stage*, sometimes called *procidentia*, the vagina is completely inside out, its anterior wall having been everted from below upwards and its posterior wall inverted from above downwards. Within the vagina lie the uterus and its appendages, the urethra, and the lower and posterior portion of the bladder. The sac may also contain small intestine, and is thus a true hernia through the pelvic floor. If the parts be replaced in their normal position, and if the patient be then asked to strain, the organs go through a definite "mechanism" which recapitulates in a moment the gradual development of complete prolapse. The anterior vaginal wall emerges first and escapes from below upwards. The cervix

suffering, and are so common, that their relief is a consideration of the utmost importance. It is well, therefore, that with the evolution of major gynecology there should have been a steady undercurrent of progress in the art and craft of plastic vaginal surgery. There are not yet enough craftsmen for the work that should be done. For every man who can cure a prolapse there are a dozen who can remove a fibroid. Unfortunately, there is still a large sale for pessaries.

Typical displacements. (1) **Prolapsus uteri** (Fig. 70).—In classical prolapse the uterus and its appendages, the bladder, urethra, and vagina are all displaced downwards and dislocated from their normal relations with the more fixed pelvic structures. Except in a few rare cases of developmental defect, the patient is a parous woman. The vaginal outlet is always enlarged, either as the result of parturition or owing to the gradual pressure exercised by the descending anterior vaginal wall.

In the *early stage* of prolapse, the bladder descends when the patient strains, and bulges at the vaginal outlet covered by the anterior vaginal wall; this is the condition known as *cystocele*. Moreover, the cervix lies farther

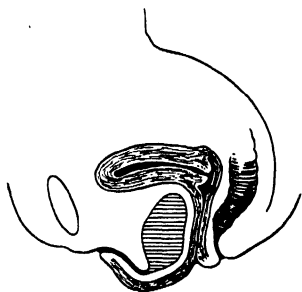


Fig. 71.—Cystocele.

follows, next comes the upper part, and, last of all, the lower part of the posterior vaginal wall.

(2) **Cystocele** (Fig. 71).—While bulging of the bladder at the vulva covered by the anterior vaginal wall is an essential feature of prolapsus uteri, it also often occurs alone. The nature of the egg-like swelling at the vulva is demonstrated by passing a sound or catheter into it through the urethra, and vaginal examination finds the uterus in its normal position of ante-

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version, the cervix looking downwards and backwards and the body of the uterus being palpable in front of the cervix. The condition occurs in parous women with enlarged vaginal outlet.

(3) **Inversion of the vagina from above downwards** (Fig. 72).—This form of prolapse often occurs in virgins and nulliparous women in

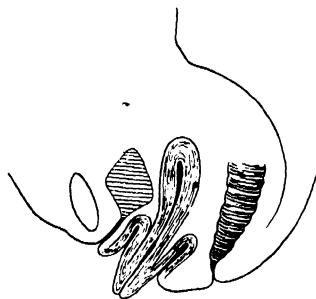


Fig. 72.—Long cervix ; inversion of vaginal wall from above downwards.

association with developmental elongation of the supravaginal cervix. When it occurs in parous women the cervix is generally split on one or both sides, in addition to being long originally. The uterus is retroverted, and its lower portion lies in the upper portion of the vagina, which is inverted from above downwards and invests the elongated cervix ; this, with both the vaginal fornices, approaches the vaginal outlet more closely than normal. With the lapse of time the condition may progress until the os uteri emerges into the vulval cleft. There is no cystocele, the cervix emerges before the vaginal wall ; and when, as occasionally happens, the condition goes on until the vagina is completely inside out, both its anterior and posterior walls are inverted from above downwards. This distinguishes the condition from prolapse in which the anterior vaginal wall is everted from below upwards.

(4) **Rectocele** (Fig. 73).—In parous women, in whom the perineum has been partially but not completely torn, the anterior rectal wall often bulges into the vulva, covered by the posterior vaginal wall. This condition is a **pouching** or local distension of the rectum with a displacement of the posterior vaginal wall. Thus it may be regarded as a form of prolapse. It occurs alone, and also as a complication

of cystocele, prolapsus and inversion of the vagina.

Genital prolapse.—This term includes the four conditions just described, namely prolapsus uteri, cystocele, inversion of the vagina (long cervix), and rectocele. It also includes prolapsus with rectocele, cystocele with rectocele, and inversion of the vagina with rectocele. Practically every case of genital prolapse falls into one or other of these groups.

(5) **Retroversion.**—The uterus is said to be retroverted when the body of it can be palpated through the posterior vaginal fornix, behind the cervix, instead of being felt in front of the cervix. This position of retroversion is common in women who complain of no pelvic symptoms and have normal pregnancies and labours. Many women with retroverted uterus have pelvic symptoms ; but many women with whom the uterus is in anteversion complain of exactly the same symptoms. Again, women often recover completely from pelvic ailments, although the uterus remains permanently in a position of retroversion. Thus the question arises whether this position of the uterus should be regarded as a displacement at all, and some authorities go so far as to hold that

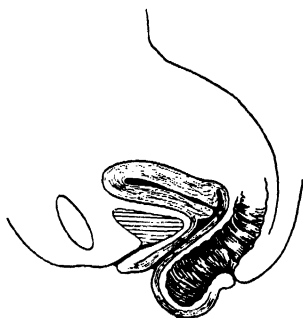


Fig. 73.—Rectocele.

uncomplicated retroversion never requires treatment. The accumulated experience of medical men shows, however, that pelvic symptoms are more common in women in whom the uterus is retroverted than in those in whom the body of the organ lies in front of the cervix. There are also cases in which symptoms disappear when the uterus is kept in a position of anteversion and return when it is allowed to become retroverted again, and this when the patient is not informed of the position of the organ at any

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particular time. Indeed, it may be taken as agreed that retroversion, in a few cases, either produces or aggravates certain symptoms, and therefore demands treatment. (For retroversion in pregnancy, see RETROVERTED GRAVID UTERUS.)

(6) **Retroflexion.**—The uterus whose long axis is concave backwards is said to be retroflexed. A retroverted uterus is often also retroflexed; but no special importance is now attached to this backward bend of the uterine axis.

(7) **Prolapse of the ovary.**—When the body of the uterus falls behind the pelvic axis in retroversion, the broad ligaments and the uterine appendages move with it backwards and downwards. Sometimes the ovaries lie protected behind the body of the uterus; but in other cases they lie, one or both of them, beside the body of the uterus in the pouch of Douglas, where they can be felt during examination through the posterior vaginal fornix. When this is the case, the ovary felt is said to be prolapsed. In this situation the ovaries are exposed to direct and painful violence during coitus. Thus the fact that an ovary lies beside the body of the uterus in the pouch of Douglas often determines the necessity for the treatment of retroversion in married women.

(8) **Anteversion.**—Until about the year 1870 the uterus was thought to be in a normal position only when its long axis coincided with that of the pelvic cavity. If the fundus were in front of the pelvic axis, the uterus was said to be anteverted, and anteversion was regarded as a displacement. Symptoms were associated with this position, and attempts were constantly made to "correct" it with pessaries. But during the last quarter of the nineteenth century it gradually became common knowledge that the uterus has no normal "position" but has rather a normal range through which it moves as the bladder fills and empties itself. The long axis of the uterus swings through an angle of about 45° in front of the pelvic axis. Thus slight anteversion is normal when the bladder is full, extreme anteversion is normal when it is empty.

(9) **Anteflexion** is a bending forward of the long axis of the uterus, and was formerly regarded as abnormal, was associated with symptoms, and treated. It is now recognized that the ordinary uterus is concave forwards, and that the curve is often very considerable in women who have no dysmenorrhœa or other symptoms and have repeated and successful

pregnancies. Modern gynecologists do not treat either anteversion or anteflexion.

(10) **Other displacements of the pelvic viscera** which are secondary to various pathological conditions are merely physical signs of those primary conditions. Thus, the pulling up of the uterus by an abdominal and the pushing of it down by a pelvic tumour are not now regarded as displacements, nor is the pulling of the uterus to one side by scar tissue or the pushing of it in any direction by an inflammatory exudate or an extravasation of blood.

Definition.—The displacements of the female pelvic viscera which demand consideration thus fall into two classes. The first includes (1) prolapsus uteri, (2) cystocele, (3) inversion of the vagina from above downwards, (4) rectocele—conditions which, with their various combinations, are often, as we have seen, included under the term "genital prolapse" or simply "prolapse." The second class includes retroversion and prolapse of the ovaries. Though they fall naturally into these two groups, all these conditions have one leading feature in common: in each there is a dislocation or alteration of relationship between the whole or some portion of the female pelvic viscera and the more fixed structures which help to occlude the outlet of the bony pelvis. For the present purpose it is convenient to consider the pelvic floor as composed of all the structures which prevent the escape of the abdominal viscera through the pelvis. The uterus and its appendages, with the bladder, vagina, and urethra, thus compose the more movable portion of the pelvic floor. The muscles of the perineal region, the pelvic diaphragm, the pelvic subperitoneal tissue, in short all the structures attached to the pelvic bones, form the more fixed portion. (Fig. 74.) *A displacement is a dislocation between the more movable portion and the more fixed portion of the pelvic floor.*

The vagina, the lower part of the bladder, and the uterine cervix are embedded in the subperitoneal pelvic tissue which intervenes between the viscera and the more fixed portions of the pelvic floor. This tissue includes no true ligaments, but the subperitoneal tissue contains a large quantity of smooth muscle which is continuous with that which forms the muscular walls of the uterus, vagina and bladder. This smooth muscle, with the firmer portion of the subperitoneal connective tissue, is arranged mainly in the form of sheaths for the blood-vessels, lymphatics and nerves which supply

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the viscera. Thus the viscera are attached on both sides by stalks of vascular and contractile tissue. So long as these lateral attachments retain their tone, the movable organs keep their position relatively to the more fixed structures of the pelvic floor. If the uterus alone becomes loose, the cervix is free to move forward, the fundus drops backwards, and retroversion occurs. If the uterus is long as well as loose, inversion of the vagina from above downwards follows. If the bladder and vagina are loose, the uterus remaining well attached, cystocele occurs. If uterus, bladder, and vagina are all loose, prolapsus or classical prolapse occurs.

Etiology.—Classical prolapse, cystocele and rectocele occur with the rarest exceptions in

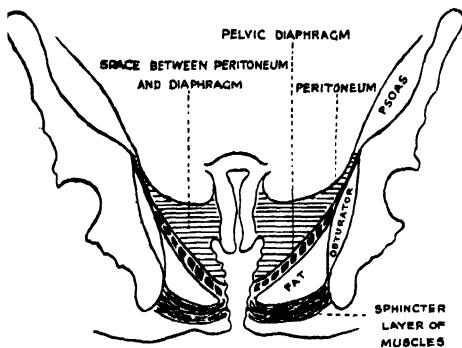


Fig. 74.—Diagram of the pelvic floor. (From the author's "Diseases of Women.")

parous women, while retroversion and inversion of the vagina are more common in parous than in nulliparous women. The main factor in the causation of dislocations of the pelvic viscera must therefore be sought in connexion with the reproductive process. The great majority of parous women, however, have no displacement whatever, though many have perineal tears. Normal pregnancy, parturition and involution cannot therefore be said to loosen the organs from their usual attachments and thus to be the cause of acquired displacements in women of normal structure. The organs are attached to the more fixed portions of the pelvic floor by the subperitoneal tissue, and this contains a quantity of smooth muscle continuous with the muscular walls of the organs themselves. During pregnancy all the pelvic smooth muscle shares in the growth of the

uterus, and during the puerperium it shares in the process of involution. Excessive or pathological involution of the uterus is well known under the name of superinvolution, and there is no reason why the name should not include excessive post-partum atrophy of the smooth muscle which intervenes between the pelvic viscera and the more fixed portions of the pelvic floor. Superinvolution may therefore be regarded as the main cause of loosening and of subsequent displacements of the pelvic viscera. But it has not hitherto been shown why this excessive postpartum atrophy of pelvic smooth muscle should occur in one woman and not in another. In short, the cause of the majority of displacements is not known.

In certain cases displacements are due to developmental errors. Thus, there are examples of complete prolapse in association with spina bifida and split pelvis. Congenital retroversion is by no means rare, for cases are often seen in which the anterior vaginal wall is short and the vagina small, the cervix being near its orifice and the small uterus being retroverted. Patients with these slight mal-developments may have normal reproductive functions; but they often begin to menstruate late, have spasmodic dysmenorrhœa, are sterile and reach the menopause at an early age.

Elongation of the cervix is a developmental error which leads to inversion of the vagina from above downwards. The long cervix lies in the vaginal axis, and thus tilts the fundus back into retroversion. If the uterus is loose as well as long it is then gradually driven like a wedge down the vagina, carrying downwards with it the inverted upper portion of the vaginal walls.

Rectocele has a separate and very definite causation, namely septic infection of perineal tears. If a perineal laceration is followed by cellulitis in the areolar tissue between the anterior rectal wall and the posterior vaginal wall, the inflammatory process leaves the rectal wall adherent to the vaginal wall instead of free to move over it. Subsequent straining at stool causes the recto-vaginal septum to bulge at the widened vaginal outlet. A patient whose perineum is torn through the sphincter into the anal canal does not strain at stool and thus does not develop rectocele.

Various fortuitous circumstances are favourable to the development of displacements, both in women whose pelvic organs have been loosened by superinvolution and in women

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who have been born with a tendency to loose pelvic viscera.

When the organs are loose, it is intra-abdominal pressure which dislocates them from their usual position. Thus, too great or too frequent raising of intra-abdominal pressure, though it does not loosen the organs in a sound pelvis, favours their displacement when they are once loose. Coughing, straining at stool, and excessive muscular exertion are therefore favourable to the development of incipient genital prolapse.

Injuries to the more fixed portion of the pelvic floor also favour the development of displacements. Thus, tearing of the perineum widens the vaginal outlet and straightens the canal. A loose uterus descends more easily and quickly if the vagina is wide and straight than if it is narrow and curved. But tearing of the perineum does not loosen the uterus, for the accident is infinitely more common than prolapse. Thus it is clear that without the essential lesion—loosening of the pelvic viscera—injuries to the more fixed portion of the pelvic floor do not cause displacements. Nor do increased size and weight of the uterus greatly favour displacement, for many large, heavy uteri are observed in perfect position, while many light and small ones are badly displaced.

Pathology.—In cases of acquired displacement the constant pathological change is atrophy of the muscular elements in the sub-peritoneal tissue which intervenes between the pelvic organs and the more fixed portions of the pelvic floor. The parametric tissues are thinned and elongated. In prolapse the pouch of Douglas generally extends farther than usual between the anterior rectal and the posterior vaginal wall. As secondary changes, the retroverted uterus and appendages are often enlarged by congestion and oedema. The prolapsed vaginal walls are often excoriated, ulcerated, thickened and skin-like. Infection of ulcerated surfaces occasionally leads to pelvic peritonitis with adhesions and occasionally with suppuration within the pelvis. There are very few records of post-mortem examinations of displaced pelvic organs. What is known of the morbid anatomy of displacements has principally been observed during operations, and from microscopic sections of portions of parametric tissue removed during operations.

Symptoms.—Patients suffering from any variety of genital prolapse generally complain of "falling of the womb," and say they have

discomfort while sitting, and difficulty in getting about and in emptying the bladder. The main complaints are often due to excoriation and ulceration of the prolapsed structures and of the vulva and thighs.

Patients in whom the uterus is found to be retroverted may have a great variety of gynecological symptoms which have no connexion with the retroversion. It is probable that the symptoms which sometimes are really due to retroverted position of the uterus are dragging pain in the iliac regions, sacral back-ache, menorrhagia, congestive dysmenorrhoea and uterine leucorrhoea. Patients in whom a large retroverted uterus presses on the rectum sometimes have great difficulty at stool, and occasionally they pass blood with the motions, although they have no piles. The special symptom of prolapse of the ovaries is definite pain on coitus. This must be distinguished by careful examination from other and vaguer forms of dyspareunia.

Diagnosis.—The diagnosis of the varieties of genital prolapse and their combinations is easy, because it is positive, and is made by the recognition of definite physical signs. The patient is placed either on her back or on her side, the knees are drawn well up and the external genitals are inspected in a good light. The patient is asked to cough, strain or bear down during the inspection, and the investigation is completed by vaginal and bimanual palpation.

Prolapsus uteri, or classical prolapse.—In the early stage there is cystocele together with retroversion. The cystocele can be seen and, when the patient strains, a finger in the vagina feels that the uterus descends with its long axis in or behind the pelvic axis. In more advanced cases the anterior vaginal wall emerges first at the vulva, followed by the os uteri and cervix. In complete cases the whole vagina is inside out. After replacing the parts inside the pelvis, the patient may be asked to bear down, when the anterior vaginal wall descends first, the cervix next, and the posterior vaginal wall last.

Cystocele.—The diagnosis is made positive by passing a sound into the bladder and noting that it enters the swelling, which bulges at the vulva covered by the anterior vaginal wall. A finger in the vagina feels the cervix looking downwards and backwards, and the body of the uterus in front of it in anteversion. The uterus does not become retroverted and does not descend when the patient strains. Most exposure of the anterior vaginal wall by laxity

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tion of the perineum must not be mistaken for true cystocele.

Inversion of the vagina from above downwards.—The uterus is retroverted, and measurement with a sound shows that its cavity is elongated. The os uteri is too near the vaginal orifice, and the cervix is invested by the inverted upper portion of the vagina. There is no cystocele, and it is not the anterior vaginal wall but the cervix which first emerges at the vaginal outlet when the patient strains. The condition must be distinguished from pure elongation of the vaginal portion of the cervix, in which the uterus may remain anteverted and the vagina may retain its usual length, although the long cervix reaches the vaginal orifice.

Rectocele.—The diagnosis is made positive by passing a finger through the anal canal and noting that it enters the pouch of rectum which bulges through the vaginal outlet covered by posterior vaginal wall. It must be distinguished from the rare posterior vaginal enterocele, which is a true hernial extension from the pouch of Douglas and contains small intestine.

Retroversion.—The mere recognition of retroversion is made by feeling a rounded solid mass in the pouch of Douglas continuous with the cervix and then making sure that this mass is the body of the uterus. This assurance can be gained by passing a sound into the uterine cavity; but the use of the sound for diagnosis has largely been given up as unnecessary, unpleasant for the patient and slightly dangerous. The mere recognition of retroversion by no means completes the diagnosis. It is necessary to establish a causal relationship between the retroversion and the symptoms complained of, and this is one of the most puzzling problems which the gynaecologist has to face. Tenderness and enlargement of the uterus, with menorrhagia, leucorrhœa, sacral backache, and pain in the iliac regions, form a combination of signs and symptoms which suggest the propriety of surgical treatment. In married women, when the uterus can be placed in anteversion and kept there by a pessary of the Hodge type, this may be done for two or three months as an experiment. If the symptoms disappear when the uterus is in anteversion and reappear when it is retroverted, the conclusion is that the retroversion causes the symptoms, and surgical treatment may be advised with confidence. The presence of a prolapsed ovary in a position in which it renders coitus painful is a clear indication for surgical treatment. The presence of results of

old pelvic infection, such as adhesions which bind the uterus to the floor of the pouch of Douglas, is a complication of retroversion which demands full consideration and generally justifies operative interference.

Prognosis.—Cases of genital prolapse progress steadily from bad to worse. They are aggravated by repeated pregnancies and labours. At the menopause some cases become rapidly worse owing to the atrophic changes in the pelvic tissues. On the other hand, the deposition of fat and the shrinking of the vaginal orifice render quite comfortable women who have previously suffered considerable discomfort. Thus some patients begin to complain, and others cease to complain, of prolapse after the menopause.

The lack of exercise often causes general ill-health in women who are prevented by pelvic displacements from getting about with ease and comfort.

In complete prolapse, ulceration and infection of excoriated surfaces may lead to septic intoxication and occasionally to local and general peritonitis. The pressure of a neglected pessary on the walls of the senile shrinking vagina may also produce ulceration, and infection of ulcers thus caused has in certain cases led to fatal septicæmia.

The symptoms associated with retroversion generally disappear as the menopause approaches, but a good many of the patients who have these symptoms become neurasthenic if they are not treated with great judgment and skill.

Treatment. Surgical treatment of genital prolapse.—Since the middle of the last century very numerous devices have been suggested and tried for the relief of the varieties of prolapse by plastic operations on the vaginal walls and the cervix. The results of the earlier forms of anterior and posterior colporrhaphy were not remarkably successful, and when abdominal surgery became comparatively safe, attempts were made to cure prolapse by the abdominal route by fastening the fundus of the uterus to the anterior abdominal wall. These abdominal operations, though fashionable for a time, could not replace vaginal operations; for they had no effect on cystocele and rectocele, and they often left the cervix protruding at the vaginal orifice. Abdominal operations, at the best, can only be used as accessory to and not in place of vaginal plastic operations for prolapse, and during recent years plastic work has been developed in

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a way that has rendered accessory work by the abdominal route quite superfluous. The evolution of the surgical treatment of prolapse has not consisted in the introduction of new devices. Many of these have been tried, but the gradual modification of the classical procedures, anterior colporrhaphy, posterior colporrhaphy, perineorrhaphy, and amputation of the cervix, has led to the adoption of a rational and reliable technique which can be varied to meet the requirements of each individual case. In prolapse the surgeon removes an appropriate portion of the anterior vaginal wall, and does not spare the cervix if it is diseased or elongated. In simple cystocele he removes a smaller portion of the anterior vaginal wall. In inversion of the vagina he amputates enough of the cervix to leave a uterus of normal length, together with a varying portion of the upper part of the vagina. In completing each operation he repairs the perineum carefully in order to restore the natural curve and slope of the vagina and to reduce its orifice to the normal size. Whenever rectocele is present a suitable portion of the posterior vaginal wall is removed in addition to the repair of the perineum. Anatomical success can almost always be secured, though, needless to say, many women have pelvic symptoms which cannot be cured by the anatomical correction of displacements. Normal parturition often takes place after these operations without any subsequent recurrence. Women over 70 years of age may be operated upon with confidence. I have described and constantly employ two recent modifications in operating for prolapse. By the first of these anterior colporrhaphy is performed, after removing a broader triangle than usual of the anterior vaginal wall. The second modification consists in combining the two separate operations of anterior colporrhaphy and amputation of the cervix in one single operation. (For details of these operations, see *Amer. Journ. of Surg.*, May, 1915, and *Journ. Obstet. and Gyn. Brit. Empire*, May, 1915.)

The operations appropriate for the different varieties of genital prolapse may be briefly indicated as follows:—

Cystocele.—Anterior colporrhaphy and perineorrhaphy.

Prolapsus uteri with normal uterus and cervix.—Anterior colporrhaphy and perineorrhaphy.

Prolapsus uteri with long uterus or abnormal cervix.—Anterior colporrhaphy combined with amputation of the cervix and perineorrhaphy.

Inversion of the vagina from above downwards.—Amputation of the cervix so as to leave the uterus 3 in. long, combined with a moderate anterior colporrhaphy. Perineorrhaphy.

Rectocele.—Colpoperineorrhaphy. A considerable portion of the posterior vaginal wall should be removed in addition to the performance of perineorrhaphy.

It may confidently be stated that these plastic vaginal operations expose the patient to no risks beyond those of taking an anæsthetic and of submitting to an incision made with modern surgical precautions. The operations demand considerable technical skill, and they also require special nursing if success is to be secured. Given these requirements, the results are all that can be desired, and afford complete and permanent relief in almost all cases. Even when pregnancy and parturition follow, there is no trouble during labour. Subsequently there is recurrence of prolapse in some cases, but in others the operation stands the supreme test of parturition with complete success.

Care should be exercised in choosing the proper time for surgical interference. Thus, no operation should be done while the parts are in a septic or ulcerated condition, lest healing should be complicated by suppuration and hæmorrhage. When this occurs the result is not necessarily ruined, but it is not so good as when healing takes place by first intention.

Operations for prolapse should be done after and not just before a menstrual period, and they should be postponed until five or six months after a confinement, as the parts are too soft and vascular during the puerperal state to present good conditions for the operator. When the vaginal walls or the cervix are ulcerated, operation should be deferred until healing has been secured by rest in bed, mildly antiseptic vaginal douching, and light packing between the douches.

The after-treatment of these operations demands very careful cleansing of the parts by the nurse at regular intervals, as well as on every occasion when the patient passes water or fæces. Vaginal douching is not desirable as a routine, but when there is purulent vaginal discharge, indicating that the lines of incision have become infected, antiseptic douches may be given once or twice daily.

The bowels should be moved the second day after operation by enema or aperient, and every day subsequently. The patient should remain in bed from two to three weeks.

Palliative treatment of genital prolapse.—This is only permissible when operative treatment is (a) refused by the patient; (b) contraindicated by disease or extreme old age; (c) postponed on account of pregnancy, lactation or other temporary considerations.

Solid rubber ring pessaries are the best instruments for prolapse. They should be sterilized by boiling and should be lubricated with soap or glycerin for insertion, as contact with oily substances causes rubber to perish very rapidly. Rubber pessaries should be removed, cleaned, and reinserted frequently. New ones are generally required every three months. Their pressure in the vagina usually causes some leucorrhœal discharge, and while they are in use the vagina should be douched with a mildly antiseptic solution twice or thrice every week. The instrument must be large enough to stay inside the vagina when the patient strains at stool, but should not be so large as to exert injurious pressure on the vaginal wall, or ulceration will follow. There should be room for a finger to be passed between the side of the instrument and the vaginal wall. After the menopause, the pessaries used should be reduced in size as the patient's age advances.

If the vaginal outlet is so large that a ring pessary of suitable size will not stay in place, the patient may try a "cup-and-stem" pessary attached by rubber tubing to a belt. Or she may have a rubber ball which can be deflated and taken out every night and inflated after insertion every morning. Another method is to pack the vagina lightly with gauze or tow and then apply a perineal pad supported by a T-bandage. In some cases the pad and bandage keep the patient comfortable without any vaginal packing.

Surgical treatment of retroversion.—When the vagina is roomy and the pelvic organs are not bound down by adhesions, it is quite possible to correct a retroversion by a vaginal operation, but this is not often done. In many of the cases in which retroversion is associated with symptoms and signs indicating operative treatment there are various results of old pelvic infection. One or both tubes may be closed, one or both ovaries may be adherent, and the uterus itself may be adherent to omentum, to the rectum, or to the floor of the pouch of Douglas. Thus, in operating for retroversion it should be the rule to open the abdomen and examine the whole pelvis by inspection. Various operations in which the round ligaments are surgically utilized to

secure anteversion are now used, that of Gilliam being one of the most popular. I have for many years used the operation devised by Webster, which is simple, safe and efficient. It does not expose the patient to the risk of subsequent intestinal obstruction, it does not interfere with the growth of the uterus during subsequent pregnancies, and it causes no trouble during labour and the puerperium.

In many cases of retroversion with symptoms, careful curetting is followed by reduction in the size of the uterus and disappearance of menorrhagia, leucorrhœa, backache, and pain in the sides. In some of these cases the uterus remains retroverted, in others subsequent examination shows that the organ has returned to a position of anteversion. After curetting an enlarged retroverted uterus, I often insert a Hodge pessary before sending the patient back to bed. This is left in position for a few weeks, and after its removal the uterus is generally found to be normal in size, shape, consistency and position.

The palliative treatment of retroversion by the prolonged use of pessaries is not to be recommended, but instruments of the Hodge type may be used by way of experiment, or as temporary expedients for periods of two or three months, if they do not cause pain and if they are really successful in keeping the cervix up and back, with the fundus in front of it. The mistake of pushing a pessary into the vagina and leaving the uterus still retroverted must be carefully avoided. Instruments cannot be used when the uterus is tender or when the condition is complicated by prolapse of an ovary.

W. E. FOTHERGILL.

PELVIC PERITONITIS (*syn.* Perimetritis). **Etiology.**—Peritonitis originating in or limited to the pelvis is common in women, and until comparatively recent years was looked upon as an idiopathic affection. It is now recognized that all cases are secondary to some primary disease in the pelvis, or to some infection, such as tuberculosis, which may reach the pelvis from a focus in the abdominal cavity. Peritonitis is, then, merely a result of disease, and its presence is beneficial in that, by causing adhesions, it limits the spread of infection.

The female peritoneal cavity is in direct continuity with the exterior through the vagina, uterine cavity, and Fallopian tubes, and it is largely due to this fact that pelvic peritonitis is so common in women. An additional reason is that the genital tract

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contains many organisms in its lower part, and after delivery at any stage of pregnancy is particularly liable to septic infection.

The most frequent and most important cause is infection from below. When infective organisms reach the Fallopian tube in this way and set up inflammation, the lumen of the uterine end, which is very small, becomes occluded by swelling of the mucous membrane, and the infected secretions from the tube escape into the peritoneal cavity through the abdominal ostium and cause peritonitis. Some degree of peritonitis always quickly follows infection of the Fallopian tubes. Thus the causes of salpingitis are the chief causes of pelvic peritonitis.

Salpingitis is a frequent sequel of puerperal infection and of gonorrhœa; it may follow other infections of the vagina and uterus, but the majority of cases are directly or indirectly due to one or both of these conditions.

The gonococcus in many cases invades the tubal mucous membrane and causes a gonorrhœal salpingitis; even if this does not occur, it may be the indirect cause of salpingitis. Other organisms easily gain a footing in the cervical glands, the endometrium and other tissues damaged by gonorrhœa, and may set up salpingitis. Pelvic peritonitis may also be caused by direct extension of infection through the wall of the uterus itself; this is most frequently seen in cases of virulent puerperal infection, but it occasionally follows intra-uterine operations. When the abdominal ends of the tubes have been closed by inflammation, further attacks of pelvic peritonitis frequently occur. They are very common in cases in which the tube contains pus, and without doubt are the result of infection traversing the wall of the tube.

There are many other causes of pelvic peritonitis which together account for a large number of cases. Tuberculous peritonitis is more common in the pelvis than elsewhere because the infective material tends to gravitate into the pelvis. Ovarian cysts and cystic ovaries are easily damaged, and peritonitis may thus result. In the formation of a hæmatocele a mild form of peritonitis occurs, and later the blood may be infected from the bowel, the peritonitis becoming acute. Pelvic peritonitis follows if an inflamed appendix lies in the pelvis. It is also commonly associated with pelvic cellulitis.

Pathology.—The peritoneum is inflamed and reddened; it loses its smoothness, and

portions lying in contact with one another become adherent. When there have been repeated attacks of inflammation, or when one attack has been of long duration, the peritoneal adhesions are firm and fibrous. Serous fluid is secreted by the inflamed peritoneum, and forms small cysts bounded by adhesions; they may be minute, but it is not uncommon to find one containing several ounces of fluid.

As pelvic peritonitis commonly arises from salpingitis, there is often a collection of this kind in Douglas's pouch around the abdominal ends of the tubes.

Intraperitoneal fluid is usually absorbed as the attack of inflammation subsides, but it may persist after all other signs have gone, or may become converted into pus. When an intraperitoneal abscess forms it may even then gradually be absorbed, or may remain for some time without undergoing much change or causing serious symptoms. The great majority, however, give rise to acute illness with fever and pain, and go on increasing in size until ultimately, unless opened, they discharge into the rectum or pelvic colon, or less frequently into the vagina or bladder. In rare instances they break through into the peritoneal cavity or find an exit through the abdominal wall.

Symptoms.—The onset of an attack is marked by severe pain in the lower abdomen with raised temperature, rapid pulse and frequent vomiting. The acute pain passes off within a few hours, leaving tenderness and discomfort over the whole abdomen, but more pronounced in its lower part. The patient lies on her back with the legs drawn up to relax the abdominal muscles and relieve the pain. Intestinal distension occurs, but is usually moderate in degree. The tongue becomes furred and the bowels constipated. The abdominal wall over the affected area moves badly on respiration, and rigidity and tenderness over this area can be detected on palpation. After some days a mass can often be felt in the lower abdomen. It may be composed of matted gut and inflamed omentum or encysted intraperitoneal fluid; in the latter case the percussion note over it is dull, but in the former may be partially resonant.

On vaginal examination there may be found a swelling in Douglas's pouch bulging down the vaginal wall, or swellings in the situations of the uterine appendages which will indicate the cause of the peritonitis. Recurrent attacks of more or less severity at long or short intervals

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commonly follow the first attack, especially in cases due to salpingitis. If the attacks are severe and frequent, the woman becomes an invalid, but if mild and at long intervals, they may have very little effect on her health.

In chronic cases dysmenorrhœa, menorrhagia, backache, and discomfort in the pelvis are commonly associated with impairment of the general health and loss of flesh. The patients are incapable of much physical exercise, and over-exertion or exposure to cold is likely to provoke a fresh attack.

Diagnosis.—The diagnosis is not as a rule difficult; an attack of acute abdominal pain with rapid pulse and raised temperature, followed within a few hours by rigidity, tenderness, and poor mobility of the lower part of the abdominal wall, is almost proof of the existence of pelvic peritonitis. In investigating these cases the important point to decide is not, as a rule, whether there is peritonitis but what is causing it.

There are, however, conditions which give rise to attacks difficult to distinguish from those of pelvic peritonitis, and in cases seen some days or weeks after the first attack the difficulties may be greatly increased.

The one most commonly mistaken for pelvic peritonitis is *tubal pregnancy* with the formation of a *hematocele* by small repeated hæmorrhages. The chief points of distinction are the history of menstruation and of bleeding, possibly with the passage of a uterine cast, soon after the attack. In cases of *hematocele* the fever is usually of shorter duration and on a lower level than in peritonitis. Later, if the *hematocele* becomes infected, the signs are those of *pelvic abscess* with acute peritonitis.

Pelvic cellulitis may be mistaken for pelvic peritonitis, but cellulitis is not a painful affection and the swelling is unilateral and in the same plane as the uterus, whereas in peritonitis it is commonly bilateral and posterior to the broad ligament, or is definitely felt in Douglas's pouch. With cellulitis there is often an indurated collar around the cervix which fixes it; with peritonitis the fixation of the cervix is less, and no induration develops in this situation unless a secondary cellulitis supervenes. Cellulitis usually develops within a few days of delivery or operation, while pelvic peritonitis is deferred until later in the great majority of cases. Rectal examination is valuable in distinguishing these two conditions: in cellulitis the bowel is often surrounded by indurated tissue which may narrow its lumen; in peri-

tonitis a mass may be felt in Douglas's pouch, but the bowel is not surrounded, nor is the mass so hard.

Torsion of an ovarian cyst, of a pendunculated fibroid, or of a coil of gut causes an attack of pain like that due to peritonitis, though early in the attack the pain is probably the result of injury rather than of inflammation.

Prognosis.—An attack of pelvic peritonitis is always a serious event, and may have disastrous effects.

The underlying inflammations of the tube, ovary, and appendix are all liable to be lighted up from time to time, and to give rise in this way to further attacks. This is extremely common in the case of salpingitis, and especially so when pus has formed in the tube.

Recurrence is therefore to be expected, and with each attack more adhesions develop, and may cause difficulty in the action of the bowels, or even give rise to obstruction.

The infection from the pelvis may be so acute in the first attack that no localization occurs, and the patient may die from general peritonitis.

Repeated attacks cause ill-health, and may reduce the patient to a state of invalidism; constant backache, pelvic pain, dysmenorrhœa, and dyspareunia may prevent her from living a normal life.

Even when the attacks seem to have ceased there is a great danger that the adhesions formed may have closed the fimbriated ends of the tubes, or surrounded the ovaries in such a way as to make the patient sterile. Sterility is perhaps the most important of all the results of pelvic peritonitis, and probably causes more unhappiness than all the others put together.

Treatment. Preventive.—As most cases are due to either puerperal or gonorrhœal infection of the tubes, many could and should be prevented. Every precaution should of course be taken in conducting labour to prevent infection.

Remedial.—The treatment of pelvic peritonitis must be determined by the cause and severity of the attack.

When *appendicitis* is the cause there is no question but that removal of the appendix should be undertaken sooner or later. If the patient is seen within the first two or three days of the onset of symptoms, immediate removal of the appendix is a safe procedure, and should be advised in order to avoid the danger of the extension of the peritonitis—a common event in appendix cases. When a case resulting

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from appendicitis is seen four or five days after its onset, operation should be postponed until the attack has completely subsided, so long as the inflammation is localized and the patient's general condition is satisfactory.

When there is still evidence of spreading peritonitis a small incision should be made and drainage of the peritoneal cavity established.

In attacks due to *salpingitis* there is not the same danger of a progressive spreading peritonitis occurring, and unless the symptoms are very urgent, immediate operation should be avoided. There are two great advantages to be gained, and practically nothing to be lost, by waiting. One, that the risks to life of operation are much less in a quiescent period than during an acute attack; the other, that many attacks subside and leave the tubes functional, if not normal, and give rise to so few symptoms that operation can be avoided altogether. When operation is undertaken in the acute stage it may mean the unnecessary sacrifice of one or both of the Fallopian tubes.

At the onset of an acute attack the patient must be confined to bed and, when necessary, measures taken to combat shock. Hot-water bottles should be applied to the limbs, and a dose of brandy or a hypodermic injection of strychnine may be given. Hot fomentations to the abdominal wall are valuable in relieving pain.

With the exception of the rare cases in which immediate operation is necessary, the treatment is directed towards limiting and quieting down the inflammation. Absolute rest in bed is essential; the diet should be confined to milk for some days at least; the bowels should be emptied by the administration of an ounce of castor oil, and afterwards by a daily aperient. Intestinal distension may be relieved by a soap-and-water enema, by one also containing an ounce of turpentine to the pint, or by the rectal tube. Morphia is often necessary for the relief of pain, but should be used sparingly, the pain being relieved, if possible, by milder drugs, by hot vaginal douches, and by fomentations to the abdomen.

With this treatment the acute symptoms in most cases gradually subside. If, in spite of these measures, the symptoms do not abate and the inflammation does not become localized, operation must be undertaken.

At this acute stage opening the peritoneal cavity and establishing drainage may be all that is safe; but when the condition of the patient justifies it and the operation is not

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difficult, it is wise to remove the diseased structures that are causing the peritonitis. A sinus is very liable to persist if the original source of the inflammation is not removed, and will increase the difficulty and danger of a subsequent operation for their removal.

In those cases in which the symptoms subside, operation may still be necessary. When there is obvious enlargement of the uterine appendages it is almost certain that there will be recurrences of the inflammation, and that with each attack the ovaries will become more and more involved; consequently, if operation is delayed too long in such a case there is a danger of finding it necessary to remove the ovaries as well as the tubes.

Drainage of the abdominal cavity is seldom needed, except in cases that are operated upon in the acute stage, when the presence of an abscess or spreading peritonitis may make it necessary. When an intraperitoneal abscess forms, it is fortunately often situated in Douglas's pouch, where it may be felt bulging forward the posterior vaginal wall. In these cases the abdomen should not be opened, but the pus should be evacuated and drainage established through a transverse incision near the cervix in the posterior vaginal wall.

J. P. HEDLEY.

PELVIS, FEMALE, MEASUREMENTS OF, AND PELVIMETRY.

—The size of the female pelvis is estimated by a clinical method known as "pelvimetry," by which certain measurements between bony points are made, and the capacity of the pelvis deduced therefrom. The pelves of all primigravidae should be measured some weeks before the expected confinement, for it is only by such routine examination that certain forms of pelvic contraction can be diagnosed early enough to avoid a difficult or disastrous labour.

Pelvmetry is both external and internal or vaginal. External measurements are made by callipers, and furnish a general idea as to the size and shape of the pelvis, but an estimation of even approximate accuracy can only be obtained by internal examination. Should all the external findings be normal, it is probable that the pelvis is normal in size and shape; but any distinct departure of external measurements from the normal is an unmistakable indication for the more trustworthy internal examination.

The principal external measurements, with the

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inferences to be drawn therefrom, are as follows:

1. The *interspinous diameter* is the distance between the tips of the anterior superior spines, and is normally about 10 in. (25 cm.). Variations of 1 in. (2.5 cm.) are common.

2. The *intercristal diameter* is the maximum distance between the outer margins of the iliac crests, i.e. points about $2\frac{1}{2}$ in. (6 cm.) behind the anterior superior spines. The usual length is 11 in. (27.5 cm.).

The chief importance of these two measurements is—(i) that the difference between them should be 1 in., and not markedly less, and,

is $5\frac{1}{2}$ in. (13 cm.), and is measured from the tip of the sacrum (not the coccyx) to the pubic arch. Slight degrees of shortening are usually met with in generally-contracted pelvises, but this diameter is also diminished in the rare kyphotic and infantile (funnel-shaped) pelvises.

5. The *transverse diameter of the outlet* is estimated by measuring the distance between the inner aspects of the tuberosities of the ischia. It is difficult to measure with any accuracy, for the bony landmark is not clearly defined. The normal length is about 4 in. (10 cm.), and it should be measured by callipers. It is diminished under the same conditions as

those which lessen the antero-posterior diameter of the outlet.

6. The *posterior interspinous diameter* is the distance between the posterior superior spines, and is normally $4\frac{1}{2}$ in. (11.2 cm.). It is diminished in general contraction, severe degrees of flattening, and in those rare pelvises where one or both lateral masses of the sacrum are absent (Naegle's and Robert's pelvises respectively).

The *internal examination* has for its chief object the measurement of the true conjugate. In ordinary practice it is usual to obtain this indirectly from

the diagonal conjugate, which is the distance from the inferior angle of the pubic arch to the promontory of the sacrum. Measure this (Fig. 75) by passing two fingers into the vagina, so that the tip of the middle finger rests upon the promontory, while the radial border of the first finger comes in contact with the pubic arch. This point of contact is marked by the finger-nail of the left index-finger, and the right hand is removed from the vagina. An assistant then measures with a pair of callipers the distance between the tip of the finger that touched the promontory, and the point of contact with the pubic arch. It is only possible to measure this diameter in cases of flattened pelvis, on account of the inaccessibility of the promontory in the normal pelvis. The true conjugate is obtained by subtracting $\frac{1}{2}$ – $\frac{3}{4}$ in., the exact amount to be subtracted depending upon the depth of the symphysis and the inclination of the pelvic brim to the horizontal. This method of esti-



Fig. 75.—Measuring the diagonal conjugate.

though the absolute length may vary, the ratio should be maintained. Any approximation of these diameters to the same figure, i.e. a diminution of the difference between them, points to some degree of flattening of the pelvic brim. (ii) A distinct reduction of these diameters, while preserving their relative difference, suggests that the pelvis is of the generally-contracted variety. (iii) The intercristal diameter (when measured from the superior lips of the crest) is about twice the length of the transverse diameter of the brim.

3. The *external conjugate* is measured from the depression below the spine of the last lumbar vertebra and the upper border of the symphysis, with the subject lying on her side. It is fairly constant at $7\frac{1}{2}$ in. (18.7 cm.). Small degrees of variation indicate nothing of significance, but if this diameter is below 7 in. it suggests that the true conjugate is flattened. It is the most valuable external diameter.

4. The *antero-posterior diameter of the outlet*

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mation should give a correct measurement to within a quarter of an inch.

Other internal measurements can only be carried out by the complicated pelvimetry of Skutch, which is seldom used in this country.

A. W. BOURNE.

PEMPHIGUS.—The term pemphigus means literally a bulla or blister, but it should not be applied to every skin affection in which there are blisters. It should be reserved particularly for the affections known as *pemphigus vulgaris*, *pemphigus foliaceus*, and *pemphigus vegetans*, all probably varieties of one complaint. It is customary, too, to use the term *acute pemphigus* for certain cases which run a very acute course, but which are probably of a different etiology, and sometimes that of *congenital pemphigus* for a complaint better known as *epidermolysis bullosa*. Other bullous eruptions which have been called pemphigus or which are likely to be mistaken for pemphigus will be mentioned when dealing with the diagnosis.

PEMPHIGUS VULGARIS

A comparatively rare disease characterized by an eruption of clear, tense bullæ, widely distributed and appearing in crops during long periods; it sometimes has a fatal termination.

Etiology and pathology.—Nothing is definitely known as to the cause of this disease. It may occur in children and adults of both sexes. It is not contagious, and there is no evidence pointing to a microbic origin. By some it has been thought to be of nervous origin, by others due to a toxin or autotoxin, but there is little or no evidence to support these views. Microscopical examinations show that there is a separation by serous exudation of the layers of the epidermis at varying depths: the horny layer from the prickle-cell layer, deeper layers of the prickle-cell area, or even of the whole epidermis.

Symptoms and course.—The complaint is essentially a bullous affection. In many cases the only symptom is the appearance of clear tense bullæ, widely distributed over the skin surface, which come out in crops during a period of weeks or months. In many cases there are at first, or even throughout the illness, no constitutional symptoms and no rise of temperature, but in others the temperature rises with each successive crop of bullæ. In longstanding cases, or when the eruption is

extensive, the patient may be made ill by the discomfort or by septic absorption from secondarily infected areas. In some cases there may be troublesome itching, but this symptom is unusual.

The bullæ arise suddenly, generally without any apparent antecedent or surrounding erythema; but sometimes there is first noticed a circumscribed patch of redness upon which small clear vesicles may appear in clusters and subsequently merge into one large bullæ. The walls of the bullæ are thin, so that they may become accidentally broken by pressure, leaving a flaccid bulla or an excoriated area.

Other bullæ may become turbid owing to infection by pus cocci. The bullæ heal without leaving a scar. In cases of extensive eruption they may be so large or so closely placed that very large excoriated areas result. The disease often involves the mucous membranes of the lips and mouth and throat, and in some cases begins in these parts, and for a long time may be limited to them. In rare instances the conjunctivæ are involved. The size and number of the bullæ vary in different cases from small scattered lesions to large bullæ covering nearly the whole skin surface. The course of the disease may also vary greatly, and, mild or severe, it may be of a few weeks' or months' duration to many years.

Diagnosis.—In the presence of a bullous eruption one should not at once diagnose pemphigus. The possibility of a bullous eruption being a *streptococcal impetigo* should be considered. In young children, and in adults in hot weather or in tropical climates, a streptococcal impetigo may be widespread over the body and limbs and bullous in character, particularly when occurring as a complication of scabies. It is only after one has had an opportunity of watching the appearance of new bullæ in spite of treatment by baths and antiseptic applications that it is possible to exclude a streptococcal infection. The affection known as *pemphigus neonatorum*, a bullous eruption which may appear in an infant a few days after birth, is actually a streptococcal impetigo and not a true pemphigus. In children *lichen urticatus* may sometimes become bullous and simulate a pemphigus. It should be remembered also that iodide of potassium, and sometimes bromide of potassium, may give rise to an eruption which is bullous in its earlier stages.

Prognosis.—The majority of cases, probably about 75 per cent., get well in the course

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weeks or months under proper treatment. But relapses are common and in some cases the disease persists with remissions and exacerbations for many years. In a few cases it is rapidly fatal, and in others death may take place after several months or years. In childhood the disease is of less grave prognosis than in adults, and a fatal termination is rare. In a few instances the disease passes from a pemphigus vulgaris into a pemphigus foliaceus or a pemphigus vegetans.

Treatment.—An essential part of the treatment is to keep the lesions clean by means of baths and mild antiseptic lotions, and as much as possible to avoid small injuries. In recent cases, and in longstanding cases where the eruption is at all extensive, the patient should be kept in bed. Nothing is more conducive to recovery than cleanliness and rest in bed. Bran baths or baths of normal saline (10 oz. of common salt to a 20-gallon bath) are better than medicated baths, and the simplest greasy applications should be employed, such as almond oil, liquid vaselin, or vaselin with the addition of zinc oxide, 1 dr. to the ounce. Butter-muslin dressings, and in extensive cases butter-muslin jackets and trousers, may be worn over the ointment and removed by being soaked off in the bath.

Internally, arsenic has an undoubted influence for good in many cases. It will often be found that a case which improves only slowly under local treatment, or in which fresh lesions appear, quickly improves with small doses of arsenic, 3–5 min. of liquor arsenicalis three times daily in water, after meals. Sometimes a case will improve with intravenous injections of neosalvarsan when arsenic by the mouth has little or no effect. In severe cases opium, in small doses of the tincture, relieves the patient of discomfort, and appears even to have a retarding effect upon the eruption of fresh bullæ. Of recent years the intravenous injection of human blood, either from another patient, or the blood withdrawn from the patient's arm and reinjected (hæmautogenous injection) has been advocated. In most cases it fails, but occasionally it has a decided curative result.

PEMPHIGUS FOLIACEUS

A rare disease characterized by flaccid bullæ of universal or almost universal distribution. It is often of many years' duration. Nothing is known as to its cause. Sometimes it follows a typical pemphigus vulgaris, and sometimes the bullæ are flaccid from the first. Owing to

the scanty exudation of fluid, the bullæ are generally little evident, and only discovered on careful inspection. The whole skin surface becomes covered with scales and thin crusts, which separate into lamellæ with fissures or weeping cracks between them. Many cases are mistaken for dermatitis exfoliativa, and the bullous nature of the eruption is discovered only after the case has been long under observation. The mucous membranes of the lips and of the mouth may show extensive excoriations, and the nails become thin and furrowed. Sometimes improvement takes place for considerable periods and the lesions become limited in extent with apparently healthy areas between, though on the skin which appears healthy it may be generally found that the epidermis can be slid off by firm pressure of the finger (Nikolsky's sign). The patient may eventually be worn out by the continuous discomfort or be poisoned by septic absorption and die during an exacerbation.

The treatment is as for pemphigus vulgaris.

PEMPHIGUS VEGETANS

This also is a rare disease. It begins usually as painful excoriations in the mouth or on the lips, or in women in the vulva, followed after a time by bullæ on the skin. The bullæ break and their base becomes covered with papillomatous vegetations. They are often most abundant, and the vegetations largest, in the genital folds, the axillæ, at the bends of the elbows, or in other folds and flexures. The lesions give rise to much pain and to offensive discharges. The disease is invariably fatal, and little can be done to stay its progress. The final state of these patients is pitiable, for they become covered with painful fungating sores, discharging an offensive-smelling pus. Such cases are sometimes mistaken for syphilis with condylomata and the eruption is greatly aggravated by the administration of iodides. Until the final stages, much can be done to alleviate the patient's sufferings by careful nursing, by baths and by antiseptic dressings.

ACUTE PEMPHIGUS

A few cases of acute pemphigus have been described, generally in butchers, or as the result of a wound when cutting meat or handling hides. The patient becomes quickly covered with large, clear, tense bullæ, is feverish, develops albuminuria, vomiting, and diarrhœa, and generally dies in the course of a week to three weeks. A diplococcus has been

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cultivated by Demme and Bulloch. A few cases have recovered with large doses of quinine.

EPIDERMOLYSIS BULLOSA

Congenital pemphigus is of rare occurrence. There is an extraordinary vulnerability of the skin, so that blisters are produced by slight injuries. The condition is usually present soon after birth, and lasts through life. It is hereditary, and may affect more than one member of the same generation. The bullæ occur mostly upon parts which are the more exposed to slight injuries, particularly upon the hands and feet, the elbows and the knees; they may appear also in the mouth. They are sometimes hæmorrhagic, may become infected by pus organisms, and generally leave scars. The nails are eventually deformed or destroyed. "Epidermic cysts" may occur at the site of former lesions, in the form of small pin-point to pin-head sized opaque white granules. The general health is unaffected. The cause is unknown. Anatomically the complaint is described as a keratolysis. Patients affected with this disease are only free from lesions when carefully protected. When they occur, lesions must be carefully dressed, and pains must be taken to avoid secondary infections.

H. G. ADAMSON.

PENTOSURIA (see URINE, EXAMINATION OF).

PEPTONURIA (see URINE, EXAMINATION OF).

PERIANAL ABSCESS.—An abscess superficial to the external sphincter in the perianal region. It may result from any source of irritation round the anus. External piles may thrombose and suppurate, a fissure may allow septic organisms to enter the adjacent tissues, and general want of cleanliness may predispose to the condition. A throbbing pain is felt near the anus, and the patient has pain on defæcation or on sitting down. Examination shows a painful swelling, often red and fluctuating, near the anus. Fomentations may be applied for a day or two, but as soon as pus has been formed the abscess should be lanced freely by a cruciform or a T-shaped incision.

ZACHARY COPE.

PERIARTERITIS (see ARTERIAL DEGENERATION).

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PERICARDITIS.—Pericarditis is justly regarded as one of the most serious affections of the heart. If we exclude passive effusions, and those due to an unexplained cause as in cancer, it is a result of infection. We must, however, avoid the tendency to look upon pericarditis as a disease in itself, and realize that it is often only one result of an infection that damages the entire heart. Undoubtedly it may be the dominant one, as in pyopericardium. In rheumatism, on the other hand, though always a grave occurrence, it may be only one factor in the production of the heart disease.

There are two cardinal dangers in an attack of pericarditis, which must claim first consideration: one is the concomitant damage to the myocardium, the other the possibility of a large pericardial effusion. Of these two, the one that is peculiar to the lesion is pericardial effusion, and both on this account and because the distinction between cardiac dilatation and pericardial effusion is one of great importance in the treatment, some introductory details will first be given of the clinical evidence of a large pericardial effusion. There is obviously a grave risk incurred if by mistaking for an effusion a severe cardiac dilatation a needle is introduced into the heart itself.

Clinical signs of a large pericardial effusion.—In exceptional cases, and more particularly in childhood, there may be distinct bulging of the intercostal spaces on the left side over the præcordial area, and an undulatory impulse may be visible in the 3rd and 4th intercostal spaces. In addition to the diffuse heart-beat, the hand may detect on palpation an increased resistance over the pericardium, and percussion gives a dull wooden note the more striking by its contrast to the surrounding pulmonary resonance. This dullness extends beyond the impulse, upward into the 2nd left intercostal space or even higher, and to the right considerably beyond the right margin of the sternum, extending from above obliquely downward and outward. The cardiac outline, when mapped out upon the chest-wall, resembles that of a pear with its stalk directed upward. The rapid development of this outline, when coupled with other signs, is very suggestive of the development of an effusion. The occurrence of marked dullness in the 5th intercostal space on the right side in the cardio-hepatic angle, a sign (Rotch's) which should be sought with the patient in the orthopneæstic position, has been much discussed. It is certainly present also in cases of extreme

dilatation of the right auricle. The auscultatory sign, which, when it can be accurately observed, is in the writer's opinion the most suggestive, is the gradual disappearance of the cardiac sounds synchronously with the progressive increase in the area of præcordial dullness. When this sign is fully developed the impression given is that the heart has been lost.

The next group of signs, the *pulmonary*, is more easily demonstrated in childhood, and concerns the posterior part of the left side of the chest. Any primary lesion of the lung or pleura must first be excluded. There is impairment of the percussion note, which is sometimes tympanitic in quality, extending from below the inferior angle of the left scapula downward towards the left axilla. In the middle of this area is a band of absolute dullness reaching to the vertebral column and, according to Ewart, extending even over to the right of the middle line. This stretches from about two finger-breadths below the scapula to the same distance above the lower border of the left lung. Over this area tubular breathing, and sometimes bronchophony or ægophony, are audible, but there are no moist sounds. If the patient is placed in the genupectoral position these signs diminish, for the pulmonary collapse to which they are due is relieved by the altered position of the heart.

Peculiar attitudes may be assumed in copious pericardial effusion. In some cases, as Hirtz has emphasized, the patient leans forward with the elbows on a bed-rest and hands on the ears. This is not distinctive, for it is met with in other cases of severe heart disease, and in particular, those with extensive pericardial adhesion. In exceptional cases the genupectoral position has been adopted, and this Hirtz believes to be symptomatic of a severe effusion. The relief given to the posterior part of the lungs by these positions is obvious.

Among the rare events in severe effusion is *paralysis of the recurrent laryngeal nerves* on one or both sides. Whether the cause is direct pressure or involvement of the nerve-fibres in concurrent mediastinal inflammation, it is not easy to decide. Again, involvement of the *phrenic nerve* may cause a most distressing paroxysmal cough, pressure on the *æsophagus* severe dysphagia, and implication of the *vagus* obstinate vomiting. All these events are exceptional.

Radiography may afford conclusive proof of a pericardial effusion by the demonstration of **two shadows, a darker one of the heart and a**

less opaque one, clearly limited by the pericardial sac, due to the surrounding effusion.

These signs, together with orthopnoea, cyanosis, or livid pallor, and a rapid pulse of small volume, give us a clear picture of a large pericardial effusion. The pulsus paradoxus has been recorded in large effusions, but is not a sign of real value.

A very important aid in diagnosis is the occurrence of pericardial friction, which may remain evident even when there is much exudation. Those who are acquainted with the difficulty of the diagnosis of pyopericardium will realize the great aid that pericardial friction gives in the diagnosis of pericardial disease.

Pericardial effusion and acute cardiac dilatation.—Clear though the picture may be in a classical example of pericardial effusion, in practice the distinction from an acute and severe dilatation may cause great difficulty. For this there are several reasons. Thus, severe dilatation may produce a very wide area of præcordial dullness, and an outline closely resembling that produced by a pericardial effusion. The cardiac sounds may become feeble and faint in acute dilatation, and on the other hand may be heard distinctly when, with a large effusion collecting posteriorly, the heart is pushed forward against the chest-wall. If, together with cardiac dilatation, there is also a much thickened and adherent pericardium the diagnosis becomes almost impossible. Again, when the radiogram shows a wide shadow but does not indicate clearly the less opaque area, corresponding to an effusion, around the darker shadow of the heart, the interpretation will need as much care as the clinical signs. The pulmonary signs recorded above may also be produced by the pressure of a much enlarged heart without any accompanying effusion. If, however, we have the opportunity of studying in any particular case the march of events from an early period in the illness, we are much assisted by the constant dullness over the præcordium and the daily fading of the cardiac sounds in acute pericardial effusion.

In all cases of difficulty we have not only, then, to weigh the clinical signs, but to combine with this our knowledge of the particular tendency of the various infective processes to produce pericarditis and dilatation. Lastly, we have again to study the character of the infection when, having decided that an effusion is present, we turn to the question whether it is serous or suppurative.

Pathology.—The pathology of pericarditis

is in itself simple. The infection carried in the pericardial circulation—exception being made for cases arising by direct extension from neighbouring viscera—produces an inflammation commencing first in the subendothelial tissues of the visceral and parietal layers. If the infection is mild a mere roughening of the opposed surfaces may result; if more severe, a considerable serous effusion; if very acute, a bloodstained effusion; if severe but less acute, a fibrino-plastic or purulent exudation, as the case may be. If it is chronic but of low virulence, great thickening of the pericardial tissues, with more or less extensive adhesions, is the result.

In the severe cases the inflammation may spread to the mediastinal tissues, and in rheumatism the pleuræ are frequently found to be affected where they overlap the pericardial sac.

When there is resolution, recovery with a degree of adhesion dependent upon the extent and severity of the inflammation will result, and in three months after a severe rheumatic pericarditis, a general adhesion of some strength may occur. Another condition not very frequent, but nevertheless well recognized, is the result of a chronic relapsing inflammation. It is associated with a similar process in other serous membranes, notably the pleuræ and peritoneum, and on this account is termed multiple serositis. This condition differs from "adherent pericardium" in that, together with the occurrence of adhesion due to former attacks, there are signs of recent activity. In the writer's experience, the chief cause of this process is tuberculosis, although some cases would appear to be rheumatic, and others are of uncertain nature.

Etiology.—Many infections attack the pericardium, but the symptoms closely resemble one another, and for the purposes of this article the most important examples are the rheumatic, which illustrate all the chief points of a non-suppurative pericarditis; the pneumococcal and septic streptococcal and staphylococcal infections, which illustrate pyopericardial effusions, and multiple serositis. In renal disease apart from hydropericardium the pericarditis is usually the result of an infection, although some authorities consider that toxæmia in renal disease may produce a pericarditis, and in particular the dry form—quite apart from the presence of any infective agent. In support of this view it has been shown that ligation of both ureters in animals has been

followed by pericarditis with the associated uræmia, but it is clearly a difficult matter to exclude with certainty the possibility of a superadded infection. This variety closely resembles in its behaviour rheumatic pericarditis. Pericarditis due to malignant disease resolves itself as a rule into a study of pericardial effusion.

We may now consider the different varieties of pericarditis.

RHEUMATIC PERICARDITIS

With few exceptions, pericarditis means a severe cardiac infection. The exceptions are robust children of about 12, who sometimes develop a transient pericarditis, which appears to leave the heart almost undamaged, and is even unaccompanied by definite endocarditis.

Again, pericarditis is more frequent in a recurrent than in a first attack of rheumatism. If it occurs in a first attack in a young child it is not infrequently fatal. As would be expected, it is much more usual in childhood; in fatal rheumatism under 12 years of age some lesion of the pericardium is present in 86 per cent.

Although pericarditis may occur in a first attack without endocarditis, it is so unusual that one suspects the diagnosis. Among the post-mortem records of the Hospital for Sick Children there is none of such a condition. In addition to endocarditis, the myocardial changes described in the article MYOCARDITIS must always be remembered. There are all degrees of virulence, and also different types of virulence. The fulminating cases are unmistakable, but the dangerous relapsing persistent forms are deceitful in their course.

Possibly it is not quite realized how frequently a hæmorrhagic effusion occurs in actively virulent cases, for the fibrino-plastic type with the shaggy coating of lymph on the opposed pericardial surfaces is undoubtedly much better known. Another form, in which there is great thickening with nodular deposits in the parietal pericardium and but little exudation, also deserves thorough recognition.

As a rule, there is some effusion, but a large quantity—half a pint, for example—is unusual in childhood. It is probably correct that considerable effusions are more frequent in young adult life. We should not then act upon the diagnosis of a large pericardial effusion in rheumatism without the strongest possible clinical evidence. Adhesions of a greater or less extent are the usual results of a rheumatic pericarditis, and in those cases in which there

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adhesions are external and implicate the mediastina and surrounding pleuræ they add greatly to the gravity of the illness.

Symptomatology.—The most severe cases of pericarditis may begin with somewhat unusual symptoms such as shivering, vomiting and diarrhœa, pointing to grave infection. There may be cardiac symptoms from the first, or a sore throat followed by arthritis or chorea and then pericarditis. The temperature is usually, but not invariably, raised, and during the attack all the manifestations or rheumatism may develop, including the subcutaneous nodules. Pain over the præcordium, shortness of breath with quick respiration, and a rapidly progressive anæmia combine to give an aspect of serious illness, and are a sure indication of severe carditis. Pericarditis, however, may also occur at this stage with so few signs of urgent illness that its discovery comes as a complete surprise.

Wynter and others have directed attention to inhibition of the action of the diaphragm in the early stage of the disease, particularly in cases with an acute and very painful onset. The physician in such cases is liable to be led astray by this sign and to seek for some acute abdominal catastrophe.

In adult life the general symptoms of toxæmia are not so striking, but the local pain and dyspnoea are sometimes even more definite, and mental disturbance, consequent in part upon the realization that the heart is affected, is much more evident. Delirium is not a frequent symptom, though sometimes in children it is associated with a rapid development of chorea. It is important in such cases to exclude the possibility of salicylate poisoning. Orthopnoea is the rule in adults, and is frequent in children, but it is nevertheless surprising in some cases of general pericarditis to find the patient lying down with comfort. Cough of a most harassing character may develop in the later stages, more particularly when the pericarditis is severe but subacute. Pleuritic pain may add much to the suffering. Although the temperature is raised, the range is usually moderate, and in the last stages in the young may for some days fall below the normal line, although active lesions are present. This is a grave sign.

Pericardial friction is very frequent. In 14 first attacks and 11 acute attacks supervening upon former pericardial disease, it was present in every case; and in 34 examples of localized recent pericarditis it was detected in 22. Eighty per cent. would probably be a low

estimate of its frequency. It is therefore a sign of the utmost value, which shows itself early in the illness, although an excited action of the heart and rapid dilatation may warn us of its probable appearance some days before its actual detection. It is usually discovered by listening over the base of the heart. In severe cases the friction rapidly becomes audible over the whole pericardium, but it is quite exceptional to detect it posteriorly. The to-and-fro character of the sound is usually audible, but sometimes a systolic or diastolic sound only can be heard, and in full inspiration even this may be difficult to detect. In all such cases of difficulty the patient should be re-examined while leaning forward.

In the early stages the friction sound is usually soft, but when the process is subsiding it may be harsh and grating. Tenderness over the præcordium may require that percussion be very gentle. The most frequent course, after the detection of the friction and dilatation of the heart, is for the cardiac sounds to become fainter, and an accompanying mitral murmur due to endocarditis, which is almost invariable, may now be almost inaudible. Most probably this marks a stage of moderate effusion, but it must not be interpreted as meaning that the wide area of præcordial dullness is thus produced; it is a factor in this, but as a rule only an inconsiderable one. For a while the friction may disappear, but it need not do so even when there is a large effusion. As the pericarditis resolves, friction may again become obvious, but præcordial tenderness and pain disappear.

The pulse from the first in severe cases is rapid, and its tension low. A progressive increase in rapidity is a serious sign, and a striking diminution in the volume is suggestive of a considerable effusion.

The course of rheumatic pericarditis is very uncertain, and not unlikely to be marked by periods of remission and exacerbation for which we can find no explanation other than that the foci of inflammation in rheumatism appear to complete their cycle of changes at different rates. It does not follow that several recrudescences will necessarily produce a grave condition, for the true gravity of the acute cases lies in the destructive myocardial changes which may prove fatal without a prolonged pericarditis. Increasing anæmia, lividity, a subnormal temperature, vomiting and restlessness, a rapid, feeble pulse, with a wide area of præcordial dullness, œdema of the face and lungs, enlargement of the liver, moderate

dropsy and albuminuria, are indications of a fatal event which may ultimately be sudden.

The severe cases of the more chronic type are recognized by the persistence of fever, great enlargement of the heart, continual pericardial friction, and signs of failure in compensation. Such cases may run a prolonged course of many weeks or even months, but long before death actually occurs it is apparent that the heart is hopelessly damaged. The writer looks upon some of these cases as examples of a malignant pericarditis comparable to the rheumatic form of malignant endocarditis, and the condition of the pericardial tissue after death affords palpable evidence of the severity of the local disease.

The existence of previous valvular disease in pericarditis is always important, because the damage to the myocardium during the attack may upset completely a previous compensation. One of the most striking proofs of this is the supervention of repeated attacks of anginal pain after acute pericarditis.

Diagnosis.—The diagnosis of rheumatic pericarditis is not as a rule difficult, on account of the frequency of pericardial friction. In some cases this sound at first may simulate closely that produced by a to-and-fro aortic murmur, but it is only a passing resemblance, and is soon distinguished by its superficial character and general distribution. If the anterior part of the pericardium is adherent and the inflammation posterior only, no friction may be detected. Undoubtedly, then, we find from time to time at necropsies undetected pericarditis, but the practical importance of this is much diminished by the fact that in such cases it has been recognized that there has been acute myocardial failure, and the question whether or not the pericardium has been also implicated has had no practical bearing upon the treatment, unless a large effusion has been overlooked.

The distinctions between acute pericarditis with much effusion and dilatation have been already considered (p. 510).

Prognosis.—Pericarditis is the lesion that most frequently coincides with fatal rheumatism, and it is almost invariable in the fatal first attacks in childhood. In 250 fatal cases of rheumatism under 12 years of age, 24 per cent. were apparently first attacks, and all had acute pericarditis.

In a series of 100 fatal cases in which 22 were first attacks, 16 occurred under 7 years of age. In 100 cases over 12 years of age, only one fatal

case over the age of 20 was recorded from acute pericarditis. In general terms, the younger the child the graver the prognosis as to life. Severe chorea is a dangerous complication.

The prognosis as to the patient's future must depend entirely upon the signs of failure of compensation that date from the illness, and in each case we have to weigh the evidence as to the cause of this failure. Is it primarily myocardial, is it the combination of valvular disease and increased myocardial weakness, or is it the added difficulty of external pericardial adhesions?

Again in general terms, the occurrence of pericarditis marks a downward step in the patient's general health. There are, however, exceptions which declare themselves by the usual signs of recovery of good compensation, and we should avoid taking a gloomy view of any case until the facts force this attitude upon us.

Treatment.—The general principles of treatment are identical with those for rheumatic endocarditis (*see* ENDOCARDITIS). In addition, there are special indications which require consideration. The first of these are local measures for controlling pain, among the most important of which are the ice-bag, leeches, blistering, and hot applications. With skilled nursing and a child or adult of ordinary strength I favour the ice-bag used continuously with all the usual precautions. This gives great relief to pain and, when it is properly adjusted and the symptoms are watched by competent nurses, is usually well borne. Undoubtedly, adults are more tolerant of the cold and consequent discomfort which at first result than are children. Lees believed the ice-bag to have, in addition, some curative action. Leeches also relieve the pain, and, when there is clear evidence of dilatation of the right side of the heart, have an additional value in the withdrawal of blood. Five may be applied to the præcordium. Both leeching and blistering make the exact examination of the heart rather more difficult. The clearest indication for blistering would be the diagnosis of a considerable serous effusion of some duration, the result of a mild general pericarditis. It seems very unlikely that a fibrino-plastic exudation would be influenced. In delicate patients, warm applications of the antiphlogistine type or light poultices are often the safest and most comforting.

Internal remedies are often needed in addition to these measures, and morphia and preparations of opium are invaluable at all ages for the

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relief of severe pain, restlessness, and mental distress. With these the bromides can frequently be combined with advantage. Chloral for insomnia is not in the writer's experience unsafe in childhood when given with care. The intravenous injection of collargol or electrargol has been advocated, but the evidence of their value is not convincing.

The surgical treatment of effusion is another special point. I have seen opening of the pericardium undertaken in the acute stage before any considerable amount of effusion has been diagnosed. In every case this diagnosis was verified by the liberation of some quantity of fluid, and recovery followed. Nevertheless, I am not convinced that recovery was obviously assisted, and such operations are fraught with anxiety. Were it possible to obtain from the exudation some specific vaccine of proved value, the risk might be worth taking, but at present no such vaccine has been obtained. When all signs point to a large effusion and its liberation is urgent, I am in favour of incision rather than paracentesis of the pericardium, on account of the fibrino-plastic character of the effusion, unless the patient is too ill for the more serious operation. The value of salicylate of sodium is considered in the article RHEUMATISM, ACUTE. I repeat here my own view upon this point, which is to employ moderate doses for the relief of painful arthritis or fibrositis, and not to push the drug with the intention of obtaining a massive "specific" effect.

In the use of digitalis or strophanthus, it is important, particularly in cases in childhood, not to disturb the digestion. These drugs have little effective value when the myocardium is acutely injured, and vomiting, once started, may mark a downward step. There are exceptions, as for example when the acute process has subsided and yet the heart requires some tonic effect; cautious trial may then show that the drug is well tolerated. Strychnine is helpful, but is not indicated when the action of the heart is much excited. Some of the best recoveries from pericarditis are the result of excellent nursing, the gentlest remedies, and the least disturbance of the patients.

I have treated ten cases of pericarditis with polyvalent antistreptococcal serum, and five with an anti-rheumatic serum, during the last fifteen years, but the results were quite inconclusive. Efforts have been made with camphorated oil, liquid paraffin, and sterilized air to prevent adhesion, but I have no experience

of these methods, and would dwell on the gravity of adhesion as largely due to external and not internal pericarditis. It must not be forgotten that camphor in large doses is highly toxic.

I advocate, especially in childhood, a freer dietary than is orthodox in rheumatism. In the acute stage patients are often very ill, and milk suitably diluted, given with or without an invalid food, is indicated, but chicken tea or mutton broth, or a mixture of the two, is also permissible if some change or stimulant effect is needed. In the later stages I am convinced that eggs, fish, chicken, if these latter be digested, are allowable, and I have never seen in childhood, the time when the most classical rheumatic affections are met with, reasonable evidence that diet has any deleterious effect upon the course of the disease. The only thing to be considered is: Can the patient, who requires generous nourishment as soon as possible, digest the solid food? Alcohol in the form of brandy or, for adults, possibly whisky is a valuable drug when the pulse is weak and the vitality low.

Convalescence must be slow, for it takes months for the inflammatory changes in the pericardium and heart-wall to heal after a severe attack. The methods are those used for endocarditis, and the principle of testing each forward step, before another advance, is identical (see ENDOCARDITIS).

It should be pointed out that some authorities are in favour of dealing with pericardial effusion at an early date when well diagnosed. In suppurative pericarditis this is clearly the correct procedure, but I advocate caution in the rheumatic cases, because cardiac dilatation is a much greater factor in this form, and also because in the majority of cases Nature deals with the effusion herself. There seems no sound reason to believe that the removal of the fluid will diminish adhesion, seeing that the worst examples of this complication are the result of a chronic pericarditis with little or no free fluid but with great damage to the pericardial tissues.

SUPPURATIVE PERICARDITIS

This provides one of the most dangerous forms of heart disease and yet one of the most difficult to detect. The frequent absence of pericardial friction and the presence of other severe intrathoracic lesions account for this difficulty.

Pneumococcal, streptococcal, and staphylo-

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coccal infections are those of chief importance, and the first of these is the most frequent.

Pericarditis may arise as an event in a general septicæmia, or may complicate an osteo-mylitis, but in these septic processes the pericardial lesion is rapidly fatal.

The *pneumococcal* form may be less virulent, though it is always a dangerous condition. It is more frequent in childhood. Early childhood especially is most liable to pyopericardium, and 84 per cent. of the cases occur under 4 years of age. The pathological processes in all forms are those of a suppurative inflammation beginning in the pericardial tissues. In childhood about 55 per cent. of the cases are associated with empyema on one or both sides, 31 per cent. with acute pneumonia and pleurisy. Although the pericardium may be affected by direct extension, it is more probable that in most cases the pericarditis arises independently. The exudation may vary from a scanty film spread over the endothelial surfaces to a large collection distending the sac; the heart does not show the dilatation met with in the rheumatic form.

The symptoms are often indeterminate. Persistent rapidity of the pulse, irregular out-breaks of fever, extreme illness, orthopnoea, dyspnoic attacks and syncope are among those most frequently recorded. Occasionally a soft evanescent friction may be audible, but the most conclusive signs are those of pericardial effusion which have been already described (p. 509). Where possible, radiography may be of the greatest value.

It is difficult to estimate the duration of the illness. Some cases run a fulminating course, others an acute course of about three or four weeks, others would seem to last for many weeks. Sudden death from syncope is frequent.

Diagnosis.—In practice the diagnostic mistakes usually made are either that the lesion is overlooked, and the symptoms are ascribed to an empyema or an unresolved pneumonia, or the vague signs lead the observer to regard acute tuberculosis as the probable cause. If the condition is part of a general septicæmia, the seat of the primary lesion—an epiphysitis, for example—may help to explain the cardiac symptoms. It is the frequent absence of pericardial friction that is so baffling to the physician, particularly as the cases are sufficiently rare to make one forget the lesson of a former failure.

The **prognosis** in very young children is gloomy, in older ones and in adults grave but

not hopeless if surgical methods are adopted. In many cases in small children, even if a correct diagnosis is made, the condition of cachexia and small amount of exudation make surgical procedures very unsatisfactory.

Treatment.—Apart from palliative measures, and the employment of vaccines with or without an initial dose of antiserum after operation, the treatment is surgical. It may be well to describe here Marfan's method of paracentesis. Marfan's puncture is made with a cannula and trocar such as is used for lumbar puncture. The tip of the ensiform cartilage is defined and the needle inserted in the middle line, directly below it. The needle is then passed upwards, grazing the under surface for about $\frac{1}{2}$ in., thus avoiding the peritoneum, and finally obliquely backward through the subperitoneal tissue, between the sternal attachments of the diaphragm, into the extrapericardial tissue, finally entering the base of the pericardium as it rests upon the diaphragm.

Blechmann points out two contraindications to this route: (1) much abdominal distension in the epigastric region, (2) considerable thoracic deformity.

West advocates paracentesis below and external to the impulse, after defining the præcordial dullness, which in these cases extends beyond the impulse. Another site for paracentesis commonly used is the 4th or 5th left intercostal space just external to the left margin of the sternum.

Paracentesis when the diagnosis is doubtful is a serious undertaking, as fatal cases from cardiac puncture bear witness.

TUBERCULOUS PERICARDITIS

Tuberculous pericarditis is infrequent. It is sometimes a "dry" pericarditis, whilst in other cases there may be a large effusion which is sometimes clear, sometimes bloodstained, and is remarkable for its tendency to recur after paracentesis has been done or even repeated. The fluid obtained is usually sterile. During the illness there may be considerable irregular fever, and the physical signs are those which have been already described in the rheumatic form, and in the section on pericardial effusion. Enormous effusions may occur and recur. Valvular diseases are not so frequent in this form of pericarditis as in rheumatism.

A condition sometimes met with in tuberculosis of the pericardium is that of *multiple serositis*. This syndrome is considered as a whole in **POLYORRHEMENTIS**. As affecting

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more particularly the pericardium, it may be described in three stages. The *first* is marked by an attack of pericarditis which may easily be mistaken for a rheumatic infection. The illness is protracted, and during this stage considerable and massive adhesions may form. The multiple character of the infection may also show itself by an attack of pleurisy on one or other side, which also runs a tedious course. The *second stage* is now entered upon, and may last for months or years. During this period fresh attacks of pleurisy or pericarditis, or even some peritonitis, may occur, and the heart, slowly strangled, begins to cause the symptoms which mark this stage.

The victims, who are usually children, are noticed to be very short of breath on exertion, and may have syncopal attacks. When fully developed, the clinical picture is very definite. The pulse is small and feeble, and is much affected by exertion. The cardiac impulse is feeble and not even palpable; the cardiac dullness is unusually resistant and extends upward into the 2nd left intercostal space, outward to or even beyond the nipple line, and rightward some distance beyond the sternal margin. The chest-wall may show systolic recession over the 3rd, 4th, and 5th left intercostal spaces. Auscultation finds the heart-sounds distant and the first sound short or the rhythm galloping. There may be no cardiac murmur at all. The veins in the neck may be full, and the pulsus paradoxus detected. Examination of the chest may show evidence of previous pleurisy. The liver and spleen are enlarged, the liver in particular. There are ascites and œdema of the extremities, and both may at first be greatly dependent upon the position of the patient. If in addition to all these signs there are evidences of local outbreaks of pericarditis or pleurisy, the diagnosis is certain.

The ascites tends to increase, and repeated paracentesis of the abdomen is needed with gradual loss in health and strength as a result.

The *third stage* is marked by the appearance of obscure nervous symptoms; headache, vomiting, irritability, nerve palsies, and possibly convulsions develop, and coma and death follow. The explanation of these symptoms is the development of tuberculous meningitis. If this complication does not arise, death results from gradual cardiac failure. The ascites may in part be due to tuberculous peritonitis.

Diagnosis.—This is not an easy matter when other tuberculous lesions are not definite. The history of tuberculosis in the family and

the absence of any evidence of rheumatism are important, but primary cardiac rheumatism is more frequent in the young than tuberculous pericarditis, and to this diagnosis one naturally inclines. It is quite possible that the dry form is more often mistaken for the rheumatic than we expect. Von Pirquet's reaction is of some assistance, and the absence of valvular disease is in favour of tuberculous pericarditis. When there is copious effusion and the condition not very acute, but the effusion blood-stained, the lesion is probably tuberculous. Discovery of the tubercle bacillus is the vital test, and I have met with an example in a young soldier in which many of the bacilli were present in the fluid; this is exceptional, and animal experiment may be needed to complete the proof. Recurrence of effusion is greatly in favour of pericardial tuberculosis.

In multiple serositis the diagnosis is often very difficult, and experience shows that most of the mistakes arise by giving the hepatic enlargement too prominent a place. Primary growths in the liver, adrenal tumours pushing forward the liver, some primary blood disease, and visceral syphilis are errors that have all occurred in the writer's experience. A more academic error is that of adherent pericardium. It is clear that this is a great factor, but such a diagnosis loses sight of the irregular progressive course and the subacute attacks of active disease in various serous membranes which mark the illness. The diagnosis rests upon a careful collection of the evidence put forward in the clinical description, and upon the exclusion of the alternatives by sifting all evidence that can be brought for and against their acceptance.

The **prognosis** is gloomy in all but the fleeting cases of dry pericarditis, though life may be prolonged for some years.

Treatment is on the general lines recognized for the tuberculous, combined with the special indications for pericarditis, including the operation of paracentesis pericardii when the effusion is copious. This may need to be repeated when the indications of a large effusion return.

The treatment of multiple serositis is palliative. Brauer's operation of cardiolysis may, however, prolong life in rare cases. In one of my cases in which this was done, health was greatly improved; five years later the patient was still living. When he was last seen he was, however, slowly losing ground from a reappearance of œdema, ascites, and enlargement of the liver and spleen. In one case the Talma-

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Morison operation for the recurring ascites proved a failure.

Although the most conclusive examples of multiple serositis that I have traced through every stage are those of tuberculous origin, examples undoubtedly occur in children as a result of rheumatism. In such cases death does not occur from meningitis but from increasing heart failure and general exhaustion of the vital powers.

The same phenomenon of strangling of the heart, described above, is also met with in rare cases in adult life, and among the most remarkable are those associated with chronic perihepatitis producing a thick white casing round the liver—the so-called iced liver (see PERIHEPATITIS). The etiology of these cases is most obscure, and the diagnosis by no means easy. There are the signs of a greatly enlarged liver with ascites, but the outstanding feature is the extreme cardiac weakness.

PERICARDITIS IN RENAL DISEASE

In renal disease it is recognized that not only may passive effusions occur in the pericardial sac, but also actual inflammation of the serous membrane. The passive effusions are as a rule associated with similar pleural effusions and anasarca, though in exceptional instances hydropericardium may develop independently.

The inflammatory conditions have been explained as the result of infection, or as due to the renal toxæmia. At the present time the balance of evidence points to both explanations as correct in different cases.

If the pericarditis is the result of infection, the agent may either be that which caused the original disease of the kidneys, or a secondary complication the result of lowered vitality of the patient. For example, a generalized pneumococcal infection may cause nephritis and pericarditis, or, on the other hand, a pericarditis, the result of a streptococcal infection, may arise in the last stage of an interstitial nephritis.

Pneumococci, streptococci, staphylococci, tubercle bacilli, and *B. coli* have been demonstrated in this form of pericarditis in renal disease. A toxic cause has been chiefly claimed for the dry pericarditis in the latest stages of chronic renal disease when the blood is profoundly poisoned; it is generally thought not invariably one of the terminal events. These forms of pericarditis are often met with in renal disease in patients over 30 years

of age, and Box's record of 10 per cent. is probably an average.

The chief clinical points of interest are the frequency with which the pericarditis develops insidiously without pain or fever in the toxæmic stage of chronic renal disease. The pericardial friction may be very harsh and grating, giving rise to a palpable rub, and being audible all over the back of the chest.

In all cases the prognosis must be very grave, but that recovery occurs is indubitable.

ADHERENT PERICARDIUM

In this article allusion has been made to the scar-formation which results from a severe pericarditis. The extent of this scarring varies from a few strands of fibrous tissue between the visceral and pericardial layers, to a complete adhesion of the two layers and the welding of the parietal to the chest-wall.

Some pericardial adhesion is frequent in the fatal carditis of childhood, and accordingly, whether the signs of adhesion described at the bedside are correct or not, the probability is that adhesion will be found post mortem. Instances have, however, occurred in my experience when adhesions have been confidently diagnosed during life, and yet after death not a single one has been found but the heart has been greatly enlarged. This warns us that many of the diagnostic signs are unreliable. It is clear, too, that the problem during life is a complicated one, for we have also to weigh in the balance the influence of valvular lesions and myocardial disease as factors in the cardiac failure. The impression left is that extensive external and internal adhesions must be injurious factors in heart disease, but to what degree it must be very difficult if not impossible to gauge.

Symptomatology.—Præcordial pain, palpitation, anginal pains, and shortness of breath are frequently present in these cases, but to what extent the adhesions are responsible for them is problematical, and there is no doubt that all symptoms may be latent.

Variation of the physical signs in different cases.—Enlargement and dilatation of the heart are the usual results of severe external adhesions, but in exceptional cases the heart may be strangled and the muscles atrophied. A third result, due to compression of the auricles, is a gradual and extreme dropsy.

Fixation of the apex beat is sometimes noticed when the patient is turned on to the left side. This is not diagnostic, for it may

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occur when the heart is very large and the chest small, irrespectively of any adhesions.

The impulse may be very extensive, and, if the heart is tied to the anterior chest-wall, may also be very forcible. Systolic retraction of intercostal spaces and the lower end of the sternum has frequently been observed, and in extreme cases systolic retraction of the posterolateral walls of the lower part of the thorax may occur.

A diastolic shock over the impulse is an important sign, but I think it exceptional. The præcordial dullness may be extensive, and unusually wooden in tone.

A sudden emptying of the cervical veins in diastole, if combined with evidence of pressure on the caval veins, as shown by œdema and ascites, suggests pericardial adhesions implicating in particular the auricular part of the heart.

The movements of the diaphragm may be impaired and the area of præcordial dullness not affected by deep inspiration. In children a diastolic murmur in the region of the apex beat may occur, and is liable to be interpreted as a result of mitral stenosis.

When the physical signs in a case of organic heart disease do not explain the severity of the symptoms, the possibility of pericardial adhesions should be remembered, but I feel that in such cases it is very difficult to determine whether the real explanation is not myocardial damage rather than pericardial adhesion.

Treatment.—No treatment of extensive scar tissue is known. Braun's operation of cardiolysis has met with some success, but the cases must be chosen with the greatest care, and the operation is formidable. One is always troubled in such cases by the fear that the myocardium may be more damaged than is supposed; and it is obvious that, if this is the case, even a successful freeing of the heart will only indirectly ease the weakened muscle, and the shock of the operation may nullify this advantage.

F. J. POYNTON.

PERICARDIUM, CALCIFICATION OF.

—This very rare condition, 59 cases of which were collected by A. E. Jones, is not suspected during life, and in the majority of instances no cardiac symptoms are complained of. Probably in most cases it is the result of pericarditis, especially of the tuberculous or suppurative forms, but the calcification may be continuous with that of the cardiac valves.

PERIHEPATITIS

In a case which I described a zone of calcareous material, like a rib, encircled the greater part of the heart at the auriculo-ventricular junction and, by pushing in the upper part of the wall of the left ventricle, produced a form of mitral stenosis.

FREDERICK LANGMEAD.

PERICHONDRITIS (see LARYNX, CHONDRITIS AND PERICHONDRITIS OF).

PERICOLITIS SINISTRA (see DIVERTICULITIS).

PERIHEPATITIS.—A chronic universal inflammation of the capsule of the liver and the adjoining peritoneum, excluding cases due to tuberculosis and malignant disease. Generally it merely forms part of a chronic peritonitis.

Etiology.—In one group of cases there is general serositis (polyserositis, q.v.), in which the pleuræ, pericardium, and often the mediastinum are involved. The kidneys are normal. Another group is associated with arterio-sclerosis and chronic interstitial nephritis. The disease is probably infective, and the infection may cause adhesive pericarditis (see PERICARDITIS, p. 517) and pleurisy as well as peritonitis. Some cases are syphilitic. There is a residuum of cases which are probably due to infection by some organism of low virulence.

Pathology.—The liver is usually a little smaller than normal and, with the spleen, is covered with a thick opaque white coating like sugar icing, which strips off easily and leaves a smooth surface. Small pits and shallow depressions are present on its surface. The whole peritoneum is thickened and ascites is always present in advanced cases.

Symptomatology.—At first there may be hepatic friction and pain in the right hypochondrium. The liver is seldom palpable, but if felt its edge is thick, smooth and hard. Jaundice is never caused by the capsulitis. There is marked ascites, and the fluid accumulates rapidly. The general nutrition remains surprisingly good, but towards the end there are exhaustion, wasting, and œdema of the legs. When chronic interstitial nephritis is present the symptoms of this disease are added.

Diagnosis.—*Cirrhosis of the liver* is often wrongly diagnosed in this disease. Great enlargement of the liver, jaundice, and hæmatemesis are against perihepatitis, and the presence of ascites before there are pronounced constitutional symptoms is in its favour. Cases which require tapping more than twice are

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usually due to perihepatitis. When the liver is small in *syphilitic cirrhosis*, it may be mistaken for this disease. If sugar is present in the urine three or four hours after a dose of 40 grm. of galactose, the condition is almost certain to be cirrhosis.

Prognosis.—The outlook is serious, especially when the kidneys are granular. In uncomplicated cases life may be prolonged for many months or years. The more rapidly the fluid accumulates, the more serious the prognosis.

Treatment.—No treatment has any ultimate effect. Antisyphilitic treatment must be adopted if the Wassermann reaction is positive. Saline purges and diuretics, of which the best is the compound pill of mercury, squills, and digitalis, are useful. Operations, such as that of Handley, to effect continuous drainage do not produce lasting improvement. If no operation is done, recourse must be had to repeated abdominal tapplings.

E. A. COCKAYNE.

PERIMETRITIS (see PELVIC PERITONITIS).

PERINEPHRITIS.—Inflammation in the region of the kidney, the lesion being within or without the perinephric sheath.

ACUTE PERINEPHRITIS (PERINEPHRIC ABSCESS).—This is usually a disease of adults, and occurs more frequently on the right side. It may arise spontaneously, may follow some acute septic condition such as tonsillitis, or may appear during the course of one of the acute specific fevers. Usually it is secondary to some injury or disease of the kidney or neighbouring organs. Severe laceration of the kidney is followed by the formation of a hæmatoma, often of considerable size, in the loin; this may suppurate, and it is especially liable to do so if an injury to the pelvis or ureter has allowed of the escape of urine.

Any inflammatory disease of the kidney may be followed by the formation of pus within the perinephric sheath, common examples being calculous disease, pyonephrosis, and pyelonephritis.

The commoner diseases of neighbouring organs giving rise to suppuration round the kidney, usually outside the perinephric sheath, are appendicitis, tuberculous disease of the spine and ribs, perforated or leaking duodenal ulcer on the right side, and empyema.

Three main varieties are described:—

(a) *Superior or subphrenic.*—Here the abscess

is situated high up immediately under the diaphragm, and tends to push the kidney downwards as it increases in size.

(b) *Posterior.*—In this, the commonest variety, the pus lies behind the kidney, and usually finds its way to the surface either at the outer border of the erector spinæ or through the intermuscular interval above the iliac crest.

(c) *Inferior.*—Usually arising from suppuration in a retrocolic appendix, the pus gradually extends downwards into the iliac fossa; it occasionally perforates the fascial sheath of the psoas muscle and, penetrating beneath Poupart's ligament, may actually point in the thigh.

Symptomatology.—The symptoms of this condition may be masked to a great extent by those of the primary disease. Thus, after the initial symptoms of acute appendicitis or leaking duodenal ulcer have partially subsided, a gradually increasing swelling in the loin may be detected accompanied by increased fever, sweating, wasting, and other signs of septic absorption. The swelling tends to bulge backwards into the loin and to show less definition of outline, and more tenderness, overlying muscular rigidity, and fixity, than a pyonephrosis. Some oedema of the superficial tissues of the loin is usually present, and there may be actual reddening of the skin in this situation. If the abscess be secondary to a renal lesion, some pus will be found in the urine, and it may be possible to elicit the history of a preceding renal calculus or pyonephrosis. When, on the other hand, the suppuration has followed tuberculous caries of the spine or a rib, a considerable bulging of the flank of the affected side may alone have drawn the patient's attention to the condition; it is accompanied by little or no fever, pain, or local tenderness.

Treatment.—A perinephric abscess should be explored from the loin and drained by an incision somewhat similar to that employed for exposing the kidney by the lumbar route. If the latter be the primary source of the suppuration it should be freely incised along its convex border at the same time and drained also. Nephrectomy, if it should prove necessary, is better left to a later date.

CHRONIC PERINEPHRITIS.—In this condition, practically always secondary to renal disease, the fatty capsule of the kidney, within the perinephric sheath, is converted into a dense fibrous or a tough fibro-lipomatous mass. The fibrous type is seen in longstanding cases of calculous disease and forms a thick, dense

sheath firmly adherent to the true capsule of the kidney, and constricting and crushing the latter. It gradually forms adhesions to the colon, the duodenum, peritoneum, and diaphragm, and may render nephrectomy an exceedingly difficult and dangerous operation. In these circumstances, however, it is usually possible to remove the kidney by enucleating it from within its own true capsule (subcapsular nephrectomy). The fibro-lipomatous form, in which the soft granular perinephric fat is converted into a tough lobulated mass, is met with in cases of chronic pyelonephritis or chronic interstitial nephritis, and in the latter a well-marked mass of fatty tissue is usually present, almost completely blocking up the renal sinus.

The symptoms, prognosis, and treatment are those of the primary renal disease giving rise to it.

HAROLD W. WILSON.

PERINEUM, TEARS OF (see LABOUR, MATERNAL INJURIES FOLLOWING).

PERIODIC INSANITY (see FOLIE CIRCULAIRE).

PERIODIC PARALYSIS, FAMILY.

Etiology.—Hereditv is the most important etiological factor in this disease, although sporadic cases are not unknown. Cases distributed over four generations of the same family have been observed. The condition is sometimes associated with migraine. Attacks of paralysis may begin in infancy or may develop as late as 30 years of age. Transmission of the disease takes place through both males and females, but usually some members of a family remain free from the hereditary tendency.

Pathology.—Little is known in regard to the pathological basis of this disease. Small portions of muscle have been excised and examined, but the changes which have been found in the fibres are not constant and cannot be regarded as specific. Hypertrophy, rarefaction, and vacuolation have been observed. It is generally agreed that an auto-infection of unknown origin is responsible for the symptoms, but investigations of the blood and of the excreta have not yet determined its nature. The urine has been found to contain a diminished amount of ammonia and creatin, especially just before and during an attack. A diminished excretion of urea has also been observed in some cases.

Symptomatology.—The patient develops

at irregular intervals a flaccid paralysis of the muscles of his trunk and extremities, which may vary in intensity and extent. The paralysis may last for a period of a few minutes to many hours, and during this time he is able to speak, to breathe naturally, to masticate and to swallow, although there may be some impairment of these functions. There is little disturbance of sensibility, but the patients sometimes complain of a feeling of numbness and heaviness in the affected parts, and the muscles may feel sore after the attack has passed away. All deep and superficial reflexes are lost, and the muscles give no response to galvanic or faradic currents during the attack. The sphincters are not affected, although micturition and defæcation are usually in abeyance while the attack lasts, possibly due to diminished secretion and excretion. Some dilatation of the heart may take place during the period of paralysis, and the pulse may be correspondingly weak and irregular. Attacks generally occur during a period of rest after muscular exertion; they are sometimes attributed to an over-indulgence in food.

Prognosis.—This is not a fatal disease, although sudden death during an attack has occurred. The frequency and intensity of the paroxysms of paralysis tend to diminish after middle life.

Treatment.—No efficient treatment is known, but the administration of caffeine citrate and citrate of potash at the beginning of an attack has been supposed to have an abortive effect.

E. FARQUHAR BUZZARD.

PERIODIC VOMITING (see VOMITING, CYCLICAL).

PERIOSTITIS.—Inflammation confined to the periosteum or, more commonly, associated with osteitis or osteomyelitis (q.v.) of the underlying bone. In this article inflammation affecting mainly the periosteum is considered.

Etiology.—*Acute* periostitis results from trauma, especially a kick or blow on the shin, or from organisms or toxins conveyed by the blood-stream in rheumatism, gout, gonorrhœa, and the various forms of pyæmia; or it may be due to direct spread from a neighbouring septic focus such as a wound or an infected tooth; it not uncommonly follows the extraction of a tooth.

Chronic periostitis may result from blood-infection in enteric fever, syphilis, tuberculosis, or rheumatism, or from trauma or involvement

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in the base of a simple ulcer. In certain chronic infections, especially of the lungs, multiple foci of chronic periostitis are apt to occur, especially in the small long bones of the hands and feet.

Pathology.—In the acute form the ordinary changes of inflammation are present—swelling and hyperæmia, and the overlying skin or mucous membrane is red, hot, and cedematous; the inflammation may subside, may progress to the formation of a subperiosteal abscess, or, by becoming chronic, may lead to the deposit of new bone, in the form of a localized node or a diffuse thickening. If suppuration occurs, superficial necrosis of the underlying bone is not uncommon.

Symptoms.—In *acute* periostitis the skin or mucous membrane related to the focus is red, hot, and swollen, and there is tenderness on pressure; the patient suffers neuralgic pain, intensified by movement or by passive congestion from lowering the part affected. If due to infection, there is a slight rise of temperature and pulse-rate.

In the *subacute* and *chronic* forms the pain is of a dull aching type, worse after exercise and at night when the part is warm, with exacerbations of neuralgia; a bony node or diffuse thickening is to be felt.

Diagnosis.—In the chronic varieties of periostitis the X-rays show a deposit on the surface of the bone; this is distinguished by its definite, even character from the ossification of a periosteal sarcoma, in which striæ extend through the tumour at right angles to the surface of the bone.

Treatment.—In *acute* periostitis the part is kept at rest and fomentations are applied; relief may often be given by the application of a Bier's bandage for an hour twice a day. Aspirin, in doses of 10–15 gr., relieves the pain; the diet must be light, and the bowels are kept lax. In obstinate cases an incision may be made through the inflamed periosteum, followed by boric-acid fomentations. In periostitis of the jaw following the extraction of a tooth a capsicum plaster is applied to the gum, and tincture of gelsemium 15 min. will relieve the neuralgia; incision of the muco-periosteum often gives immediate relief.

Chronic periostitis is treated by dealing with the general disease which causes it; local treatment is by counter-irritants, combined with splinting of the limb in a raised position; Scott's dressing, blisters, and leeches are useful. To promote resolution, potassium iodide is given; Bier's treatment by passive congestion

PERITONEUM, NEW GROWTHS OF

is also of value. In many cases, however, relief is only to be obtained by incision of the periosteum, and in some it is necessary to gouge a gutter in the underlying sclerosed bone.

Typhoid periostitis usually attacks the tibia, a rib, or the sternum, and causes a subperiosteal abscess which must be incised and drained; subsequent sequestrectomy may be necessary.

Tuberculous periostitis, most commonly met with in the vertebræ, ribs, sternum, and tibia, is described in the articles on SPINAL CARIES and BONE, TUBERCULOSIS OF.

Syphilitic periostitis, which occurs in the congenital and the acquired forms of the disease, and causes periosteal nodes of the long and flat bones, especially the tibiae and the skull bones, is considered in BONE, SYPHILIS OF.

C. W. GORDON BRYAN.

PERIOSTITIS, ORBITAL (see ORBIT, AFFECTIONS OF).

PERIPHERAL NEURITIS (see MULTIPLE NEURITIS).

PERISIGMOIDITIS (see DIVERTICULITIS).

PERITONEUM, NEW GROWTHS OF.

—The peritoneum, though seldom the seat of a primary malignant neoplasm (endothelioma, sarcoma), is not infrequently involved in **secondary malignant disease**. Primary growths of the stomach, intestines, gall-bladder, breast, uterus and ovaries are the more important tumours which may affect the peritoneum secondarily. The involvement may take place (1) by direct extension, (2) by permeation of lymphatics, (3) by the setting free of cancer-cells which become implanted on the peritoneum after the fashion of grafts, and possibly (4) by an embolic process. In many cases peritoneal involvement is recognized as an incident in the clinical course of a case of malignant disease of some abdominal viscus, but, in others, signs of peritoneal disease attract attention before the existence of the primary growth has been suspected. In this latter group it is common for ascites to be detected before the invasion of the peritoneum by secondary cancerous nodules becomes obvious. A careful examination of the abdomen may reveal such masses only at a later stage. Important corroborative evidence may be afforded by induration around the umbilicus, enlargement of the inguinal glands and of glands above the inner end of the left clavicle, and possibly by a tumour in the upper part of the abdomen due to cancerous

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infiltration of the omentum. If the abdomen be tapped the fluid is frequently found to be bloodstained, and microscopical examination may show the presence of malignant cells. Loss of flesh is usually progressive, and a definite cachexia develops, attended in some cases by pigmentation of the skin. Death usually occurs within a few months of the onset of ascites.

Treatment consists in alleviating symptoms. Paracentesis may be necessary to lessen respiratory embarrassment, and occasionally an operation for the relief of obstruction may be called for.

Simple new growths of the peritoneum are very rare. Unless reaching a large size, their presence is not likely to be suspected. They are sometimes discovered unexpectedly at operation or necropsy. Strictly speaking, most so-called simple tumours of the peritoneum really originate in the extraperitoneal cellular tissue. Fibromata, myomata, lymphangiomata and lipomata are the tumours chiefly met with. Some retroperitoneal *lipomata* attain an enormous size. Those originating beneath the parietal peritoneum are sometimes of special clinical interest from the fact that they may become the starting-point of a hernia. **Cysts** of the peritoneum comprise: (1) Parasitic cysts, of which the most important are hydatid cysts: these may be found in the extraperitoneal tissue or be free in the peritoneal cavity. (2) Lymphatic cysts, either serous cysts containing clear fluid or chylous cysts filled with a chyle-like material. (3) Enteric cysts, derived primarily from the intestinal tract, showing secreting epithelium and muscle-fibres in their walls: they occur for the most part between the layers of the mesentery. (4) Dermoid cysts. (5) Blood-cysts. (6) Cysts containing soft caseous material derived from tuberculous mesenteric glands. (7) Papilliferous cysts secondary to rupture of a papilliferous cyst of the ovary. (8) Neoplasms which have become cystic. Laparotomy and removal when feasible is the usual treatment.

C. E. LAKIN.

PERITONITIS, ACUTE GENERAL.—

General inflammation of the peritoneum, which may be either primary or secondary. The term *primary* or *cryptogenic* is applied when no definite antecedent lesion can be discovered. In these cases it is usually impossible to determine the portal of entry of the bacteria into the body, but in most instances the organisms appear to be conveyed to the peritoneum by

the blood-stream. Primary peritonitis is of rare occurrence, and most cases are due to a pneumococcal or a streptococcal infection.

Cases of *secondary* diffuse peritonitis usually begin as a localized infection of the peritoneum, and this in turn is generally dependent upon inflammation or rupture of one or other of the abdominal viscera. Among the more frequent causal conditions are appendicitis; perforation of a gastric or duodenal ulcer; perforation of intestinal ulcers as in typhoid, dysentery, or more rarely tuberculosis; salpingitis and pyo-salpinx; cholecystitis; rupture of the urinary bladder or of the kidney. An abdominal contusion with attendant bruising of viscera, should an auto-infection be superimposed, may determine a localized peritonitis. Penetrating wounds of the abdominal wall may provide a direct means of access for organisms (exogenous peritonitis), but more frequently the resulting inflammation is to be ascribed to perforation of a bacteria-laden viscus (endogenous peritonitis). The only anatomical communication with the outer world is by means of the Fallopian tubes, and gonococci and tubercle bacilli sometimes gain an entrance by this route. More commonly organisms pass by way of the lymphatics, and instances of this are seen in puerperal infections of the uterus and in infections of the umbilical cord in the new-born. In health the passage of organisms from the intestine into the peritoneal sac is prevented by the integrity of the visceral layer of the peritoneum. When interference with the circulation of blood through a segment of bowel occurs as in strangulation of a hernia, the endothelial cells no longer form an effective barrier to the egress of organisms, and peritonitis results. Little is known of the production of peritonitis apart from the agency of bacteria. A diffuse non-suppurative peritonitis is sometimes seen after the rupture of an ovarian cyst or of a hydatid, or the traumatic rupture of a healthy gall-bladder. If such conditions are not promptly treated there is risk of injury to the serous layer covering the intestines, and with diminution in the vitality of the endothelium an endogenous infection from the intestinal tract is liable to occur.

The peritonitis which follows surgical intervention is usually the result of failure to secure a perfect anastomosis between two portions of intestine. In some cases the junction has been imperfectly effected, in others excessive suturing has produced so great a constriction of the tissues that their blood supply is interfered

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with and a consequent necrosis allows of gross contamination of the peritoneum.

Inflammatory changes in the peritoneum resemble those occurring in other serous membranes, save that the resulting exudate is more frequently purulent. The defensive powers of the peritoneum are very great, and vastly beyond such as are possessed by a serous structure like synovial membrane. The chief factors in the defensive process appear to be the phagocytic action of endothelial cells and leucocytes, the bactericidal properties of the exudate, and the absorptive powers of the peritoneum and omentum. The resistance of the peritoneum is reduced by anything producing injury to the endothelium; thus at operation special care should be taken to prevent drying of the serosa and to protect it from cold. So long as the invading bacteria are limited in number they are absorbed and carried by the lymphatics to lymph-glands, where they are destroyed; but once gross damage of endothelium has occurred, large numbers of organisms are allowed to pass freely into the general circulation, and septicæmia results. The presence of foreign bodies in the peritoneal cavity, such as bloodclot, or, in perforative cases, particles of food or faecal matter, by providing a nidus for the multiplication of bacteria, or a citadel for their protection, adds largely to the gravity of a case.

Bacteriology.—Many different organisms are concerned in the production of diffuse peritonitis. The most important are the colon bacillus, staphylococci, streptococci, pneumococci, gonococci, *B. typhosus* and *B. tuberculosis*. The colon bacillus is found most often in cases originating in lesions of the intestinal tract. It is usually accompanied by other organisms such as streptococci and staphylococci. Its virulence varies greatly. Dudgeon and Sargent believe that a white staphylococcus frequently present in peritoneal inflammatory exudates exerts a protective influence in that it gives rise to an exudate rich in phagocytes and appears to promote the formation of adhesions. Streptococci and *B. pyocyaneus* are responsible for the most virulent type of case, the patient often being rapidly overwhelmed by the severity of the toxic symptoms. In some of these cases local signs may not have time to develop and at necropsy peritoneal exudate may be absent, the only indication of a diffuse peritonitis being vascular injection of the peritoneum, with slight dulling of the serous surface. In some cases of diffuse peritonitis

the pus may be found to be sterile, but in many of these, if a culture be made by gently scraping the surface of the visceral peritoneum, organisms can be demonstrated.

Morbid anatomy.—In the more usual forms the intestines are in a state of paralytic distension, and their walls infiltrated and softened. The peritoneal coat is injected and dulled, and usually glued to a neighbouring coil by a sero-fibrinous exudate. The exudate is subject to much variation. In virulent streptococcal cases it is often thin, odourless, and sometimes bloodstained, in gonococcal cases it is dry and fibrinous, while in cases for which *B. coli* is responsible the exudate is commonly cream-like in consistence. In perforative cases particles of food or faecal material are likely to be found. The amount of effusion may vary from a few ounces or less to many pints. The omentum is often adherent at the site of the primary lesion. In some cases localized abscesses may be found in addition to the diffuse inflammatory changes in the peritoneum.

Symptomatology.—The symptoms vary with the cause of the peritonitis and with the intensity of the infection. In perforative cases there may be the sudden onset with agonizing pain followed by signs of collapse. In chronic visceral disease, on the other hand, not only may the onset of a terminal peritonitis escape notice, but its very existence may remain unsuspected during life. In cases of perforation of a gastric or duodenal ulcer, with sudden agonizing pain, examination of the abdomen shows the upper portion to be immobile, rigid, and extremely tender; after a few hours signs of collapse diminish and the patient appears, all too fallaciously, to be better. This "stage of repose" is followed by the reappearance of symptoms due to commencing peritonitis. Pain returns and becomes continuous; exacerbations occur from time to time, and the pain is increased by movement or pressure. Thus hiccough, cough, vomiting, and micturition may all be attended by severe pain. As time goes on, the pain may again disappear, but this must not be taken to indicate improvement. It is due rather to the effect of the profound toxæmia on the sensory centres. The abdomen, which in perforative cases is at first flat, hard and tense from muscular rigidity, gradually becomes distended. The distension is sometimes to be ascribed to the accumulation of gas in the bowel consequent upon the arrest of intestinal peristalsis, and in perforative cases to the presence, in addition, of a certain amount

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of free gas in the peritoneum. The legs are kept drawn up to relax the abdominal parietes, and the actively moving chest contrasts forcibly with the motionless abdomen. General rigidity in early stages of diffuse peritonitis is the rule, passing off as the end draws near, but exceptionally it is absent throughout. Exactly the same statements may be made with regard to abdominal tenderness.

Vomiting is usually a distressing symptom and is characterized by the slight effort with which the stomach contents are ejected. It is common for small quantities of fluid to regurgitate gently into the mouth. It occurs on the least provocation and is excited by taking fluid for the relief of the distressing thirst. At first the vomit consists of gastric contents, then the presence of bile shows that reflux from the duodenum is occurring. In severe cases the vomited matter is very dark from the presence of blood coming from the deeply injected stomach, but faecal vomiting is less often seen than in cases of intestinal obstruction. Constipation is the rule, and occasionally may be so severe as to suggest intestinal obstruction. An enema is likely to bring away faecal matter, but the evacuation is unattended by any improvement in the patient's condition. Diarrhoea occurs in many puerperal and pneumococcal cases.

The pulse is usually small, incompressible, and of increased frequency from the onset, and the temperature raised; where, however, the patient's resistance is feeble the temperature, unless the rectal temperature be taken, may be subnormal throughout. As the illness proceeds, the pulse becomes progressively more rapid, reaching 160 or 170 and eventually becoming uncountable and thready. In the early stages the blood-pressure is raised, but falls as the results of the toxæmia become manifest. The aspect of the patient is characteristic. The features become pinched, the eyes sunken, the expression anxious. A peculiar greyish pallor is sometimes noticeable. The tongue is dry and shrivelled. The voice becomes thin and husky, the extremities cold.

Physical examination reveals shallow respiratory movements costal in type, and a distended, motionless, rigid abdomen which is tender on palpation. Percussion elicits a hyper-resonant note over the abdomen, and possibly some dullness in the flanks due to the accumulation of fluid. Liver dullness is likely to be absent; this is often to be ascribed to gaseous distension of the intestines but may be

due to the presence of free gas in the peritoneal cavity. It should be borne in mind that the existence of adhesions between the anterior abdominal wall and the liver may prevent disappearance of the hepatic area of dullness, even though free gas be present in the peritoneal cavity. Auscultation usually shows a complete absence of gurgling sounds. Examination of the blood reveals a fairly constant leucocytosis. The urine is scanty, sometimes albuminous, and not infrequently retained.

Diagnosis.—*Acute thoracic disease* involving the diaphragm, such as a basal pleuro-pneumonia or a diaphragmatic pleurisy, is occasionally mistaken, on account of abdominal pain and rigidity, for general peritonitis. In these cases the rapidity of the respirations—40 or more a minute—suggests a thoracic rather than an abdominal location for the lesion, and if firm pressure be kept up with the palm of the hand the abdominal wall is felt to relax at the beginning of each inspiration. The rigidity of peritonitis is continuous. In thoracic cases the abdominal tenderness is superficial, and deep pressure is usually not objected to. Examination of the chest generally reveals some distinctive physical signs, but the appearance of these is sometimes delayed for twenty-four hours. The "catch" so often heard at the end of inspiration in a patient suffering from pleurisy will afford, if present, an important clue. It is to be remembered that peritonitis may supervene upon a case of *intestinal obstruction*, its onset being indicated by fever, increasing pulse-rate, abdominal rigidity, and possibly hiccough. In a pure case of intestinal obstruction the abdominal walls are flaccid, the pain is more paroxysmal and less continuous, tenderness is often absent, and constipation, once the lower bowel has emptied itself, is absolute, neither faecal material nor flatus being passed. Vomiting is more copious and, although its advent must not be awaited, faecal vomiting is earlier in obstruction. Visible peristalsis will point to obstruction. A patient suffering from *chronic lead poisoning* and showing the blue line on his gums may present himself with acute abdominal pain and constipation. Such a case of colic will be distinguished from peritonitis by the pain being more paroxysmal and less continuous, by its being relieved by pressure, and by the absence of vomiting. Should definite tenderness be present in colic, it may be very difficult to arrive at a diagnosis, but the characteristic pulse of peritonitis is absent. In *hysteria* a condition of "peritonism" may be met with,

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and considerable difficulty is frequently experienced in distinguishing it from a true peritonitis. Inquiry should be made as to the occurrence of similar attacks in the past. Though superficial hyperæsthesia is present, deep pressure may often be exerted, provided the patient's attention is diverted, without eliciting tenderness.

Prognosis.—When the infection has become generalized throughout the peritoneal cavity, very few cases recover. Where operation is possible, the earlier it is undertaken the better is the prognosis. The outlook after operation is more favourable in bullet wounds or traumatic rupture of the intestine than in perforation due to disease. The prognosis is better in perforation of a gastric or duodenal ulcer than of ulcers lower down the intestinal tract; this appears to be due to the fact that the intestinal flora progressively increases in profusion as the ileo-cæcal valve is approached. Gonococcal cases are often comparatively mild, though they may be fatal. In Dudgeon and Sargent's series of cases pure *B. coli* infections proved more deadly than those in which the white staphylococcus was present in addition. Streptococcal cases, if generalized, are almost always fatal. Absence of leucocytosis is a bad omen.

Treatment.—An attempt to trace the source of the infection is to be made in all cases. Where it is possible to deal with the condition surgically, no time should be lost. In perforative cases the earlier the operation the better the patient's chance of recovery. Drainage, with as little disturbance as possible in removing gross contamination from the peritoneum, is all that is usually necessary. The surgeon is careful not to break down barriers which are being formed against the spread of the infection, and attempts at an elaborate toilette of the peritoneum are usually ill-judged. The Fowler upright position should be assumed as soon as possible after operation. Good results sometimes follow the introduction of large quantities of fluid into the body. It is suggested that not only are toxins already in the blood diluted, and their excretion through the kidney hastened, but, by filling the vascular system, the current of absorption, which normally passes from the great serous sacs like the peritoneum into the blood-stream, is reversed. In other words, instead of toxins passing into the blood from the infected peritoneum, the tendency will be for fluid (lymph) to pass out from the blood-stream into the peritoneal cavity. When a drainage

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operation has been performed a sort of physiological irrigation, it is suggested, is in this way established. Fluid may be introduced, either by intravenous saline infusion, by subcutaneous injection, or by proctocolysis (the instillation of fluid into the rectum). If the latter method of administration be adopted, it is possible for 12-16 pints of water to be absorbed in the twenty-four hours. A tube provided with several openings is passed into the rectum and connected with a reservoir containing normal saline or water at a temperature of 105° or 110° F. The reservoir is kept about 2 ft. above the level of the bed and the fluid allowed to run in slowly. Discretion must be exercised as to the amount administered; if too much is given, waterlogging of the lungs and other tissues may occur. Vomiting may be relieved by small repeated doses of tincture of iodine, but the most effective measure for its relief is lavage. In some cases it may be wise to keep a small tube in position, so that lavage can be performed at frequent intervals without greatly disturbing the patient. Tympanites may be relieved by using the rectal tube. Purgatives must be avoided, for increased peristalsis favours the spread of infection. Opium is best withheld, except in hopeless cases to relieve pain and suffering. It increases intestinal paralysis and inhibits phagocytosis.

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PERITONITIS, CHRONIC.—This term is employed to include several different clinical types. It is often wrongly applied to the changes which result from a previous attack of acute peritonitis, when for instance intestinal coils are inextricably bound together by adhesions or the peritoneal cavity is obliterated. It should be restricted to chronic, progressive inflammatory conditions of the peritoneum. By far the larger group consists of cases in which chronic inflammatory changes are associated with a tuberculous or a cancerous invasion of the peritoneum (*see PERITONITIS, TUBERCULOUS*). Another form is that known as *proliferative peritonitis*. This appears to be due to infection of the peritoneum by organisms of low virulence; and depressing influences such as chronic alcoholism, lead poisoning, syphilis, or chronic Bright's disease may predispose. The actual lesions vary. In one variety the peritoneum is beset with innumerable minute nodular thickenings and opacities which may easily be mistaken for tubercles; these are formed by the local pro-

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liferation of endothelial and connective-tissue cells, but no central caseous spot is to be seen. In another variety the proliferative change, when advanced, is lamellar rather than nodular, and layers of dense fibrous tissue cover the viscera. Adhesions may or may not be present. The liver and spleen are usually enclosed in a dense white covering, compared, not inaptly, to a coating of icing sugar. In certain of these cases the liver is cirrhotic to some degree and ascites is frequent. In another group of cases of chronic peritonitis the peritoneal effusion is part of a *polyserositis* (polyrrhomenitis), or associated with chronic inflammatory changes in the mediastinum - *chronic mediastinitis*. Lastly, cases of chronic *hamorrhagic peritonitis* are occasionally encountered. No matter which variety be present, the bowel usually shows distortion and kinking, and its outer peritoneal coat is thickened, while in places its muscular coat is thinned and atrophied. Here and there the intestine is commonly bound down by fibrous bands and membranes, and multiple adhesions are frequently present.

Symptomatology. — The symptoms and signs vary in the different forms and are largely to be explained by the mechanical conditions obtaining within the abdomen. When chronic peritonitis complicates chronic visceral disease, such as cirrhosis of the liver, ascites is the predominant feature. Some of these patients live to be tapped a score or more times, a peculiarity which contrasts forcibly with what is experienced in cases of ascites of purely hepatic origin. In many cases of chronic peritonitis the symptoms are so indefinite that diagnosis may long be dubious and only be arrived at eventually by a process of exclusion. More attention should be paid to signs than to symptoms, yet there are cases in which signs are scarcely appreciable. If effusion is absent the abdomen is often flattened and retracted, but meteorism is by no means infrequent. The distension, which is rarely quite uniform unless a considerable effusion is present, varies from time to time. Visible peristalsis is sometimes noticed. On palpation, much unevenness can be detected, and a peculiar doughy sensation is often imparted to the hand. Occasionally friction may be felt over the abdomen. Nodular masses are not infrequent, especially in tuberculous cases. On account of the shortening of the mesentery, it may be impossible to demonstrate alternating dullness, even when considerable ascites is present. Percussion may reveal

a curious lack of uniformity from day to day. This is due partly to changes in position of accumulations of gas within the intestines, and partly to intraperitoneal collections of fluid being shifted from place to place by peristaltic action.

Pain is the commonest symptom. In character it may be colicky or dragging. It is aggravated by movement, and the patient often complains that he feels it most when walking downstairs. It is frequently relieved by the pressure of a binder, but in some cases no pressure can be tolerated, so great is the tenderness. The appetite is poor, and the taking of food may be followed by pain. This is probably to be ascribed to the action of adhesions in impeding orderly peristalsis. Constipation is usually present, and painful micturition and dysmenorrhœa may be complained of. Apart from these local manifestations, the patient complains of progressive weakness and loss of weight. He presents a sallow appearance, and becomes depressed and irritable. From time to time he is subject to attacks of fever, and in some cases there are associated symptoms due to involvement of the pleura and pericardium.

Diagnosis. — This is frequently difficult, from the great variability in the symptoms and signs. When abdominal pain is the sole complaint, if such pain arises in the course of digestion or follows upon defæcation, the possible existence of chronic peritonitis should at least be borne in mind. The lumps sometimes felt on palpation of the abdomen, produced by inflammatory thickening and bowel entanglement, may be mistaken for malignant disease, and in some cases laparotomy may be the only means of deciding the point.

Prognosis. — Serious impairment of the general health almost always occurs. The course is usually a gradual downward one. Sometimes the symptoms abate, only to reappear later. Death is usually due to asthenia or to some intercurrent infection, e.g. pneumonia. Intestinal obstruction may threaten from time to time and finally become definitely established.

Treatment. — If extensive effusion exists, paracentesis is indicated, and the operation may have to be repeated from time to time. Injection of 1 or 2 dr. of 1-in-1,000 adrenalin solution before the cannula is withdrawn may delay recurrence of the effusion. Applications to the abdomen with the object of promoting absorption are frequently employed. In an

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adult, 1 dr. of ung. hydrarg. oleati may be rubbed over the abdomen daily so long as no signs of mercurialism become evident or no harm to the skin results. Tr. iodi mitis applied two or three times a week is another favourite application. Hot fomentations, or turpentine stupes, or glycerinum belladonnæ (B.P.C.) may be applied if there is much pain, and a binder often gives relief. Food yielding as little residue as possible is preferable, such as milk, eggs, cream, and meat; while sunlight, fresh air, and tonics such as cod-liver oil, malt, and iron will serve to maintain the general health. Intestinal obstruction may call for surgical intervention.

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PERITONITIS, PNEUMOCOCCAL.

Etiology.—This disease is met with more commonly in children than in adults. Girls are affected more often than boys. It is not infrequently *primary* or *cryptogenic*, no other focus of infection being discoverable. There is little doubt that these so-called primary cases are really septicæmic in origin. In other instances a definite path of infection may be disclosed. These paths of infection include (1) the Fallopian tubes; (2) the alimentary tract, especially the vermiform appendix; (3) lymphatic communications between the pleura and peritoneum, e.g. peritonitis secondary to a pneumococcal pleurisy; (4) the blood-stream, the organisms being carried from a pneumococcal focus in the lung, middle ear, etc. Or the pneumococcal peritonitis may be an event in the course of a general pneumococcal septicæmia in which the meninges, pericardium, and synovial membranes may all participate.

Pathology.—The infection may culminate in a *circumscribed* inflammation with abscess-formation, or in *diffuse* peritonitis. No essential difference in the pathology of the two forms exists, though their clinical aspects are dissimilar. The circumscribed form represents a process which is less virulent or more vigorously resisted.

The inflammatory exudate poured out in response to a pneumococcal infection is peculiarly rich in fibrin—a fact that may serve to explain the frequency with which a pneumococcal peritonitis becomes localized. The exudate is usually liquid in character, greenish, and contains numerous flocculi of fibrin; in other cases it is so highly fibrinous that it forms a junket-like coagulum of a yellowish colour. Unless other organisms such as *B. coli* are present, the exudate is odourless. Con-

trasted with other peritoneal infections the limiting abscess-wall is particularly thick. The subperitoneal tissues are usually found to be oedematous, and the intestines red and injected. A bacteriological examination, however, is the only means of arriving at an exact diagnosis.

The localized form with abscess-formation usually affects the lower portion of the abdomen, and the abscess may point at the umbilicus just as in the case of a tuberculous infection, or it may rupture into the bladder or vagina. After spontaneous evacuation complete recovery may result, but not infrequently further drainage is required.

Symptomatology.—The clinical picture of a pneumococcal peritonitis is often well defined, yet the differentiation from other varieties is not easy. In the diffuse form not only is the onset acute, but the peritonitis becomes straightway generalized. In this latter respect it differs from most cases of peritonitis, which commonly start as a local infection and only later become generalized. In some cases the coexistence of pneumonia, pleurisy, or meningitis may afford an important clue.

A complaint of pain in the lower part of the abdomen is usually the first indication that something is wrong. The pain recurs at short intervals and each attack is accompanied by vomiting. The vomiting may become persistent for a time and is usually associated with diarrhoea. The temperature quickly rises to 102° or 103° F., and the pulse-rate in children is likely to reach 140 or 150. In the circumscribed form, examination of the abdomen usually reveals tenderness and rigidity in one or other iliac fossa. Appendicitis may be closely simulated, but rigidity and tenderness are rarely so limited in extent as in cases of appendicitis. In the diffuse form tenderness may be more generalized, movement poor, and evidence of constitutional toxæmia more profound. At first there is often no distension. To explain this it has been held that pneumococcal infections are less toxic than other pyogenic infections to the intestinal musculature, and that therefore paresis of the bowel with its associated distension and constipation is slower in appearing. Percussion may show the presence of fluid in the flanks and across the pubes within twenty-four hours of the onset. As the patient becomes worse the abdomen is increasingly distended, the mouth covered with sordes, the breath fœtid, and herpes may appear on the lips. The face, at first flushed,

becomes dull and listless, and the eyes sunken. Death often occurs within two or three days from toxæmia, or may be deferred to the sixth or seventh day. A few cases recover completely. Occasionally the illness is prolonged, and residual abscesses form from time to time.

The main difference between the circumscribed and the diffuse type is to be ascribed to the greater area of peritoneal involvement in the latter and the increased toxic absorption which results.

Diagnosis.—The *diffuse* type has sometimes been mistaken for typhoid fever on account of the association of diarrhœa with profound constitutional disturbance. The features which differentiate pneumococcic peritonitis are the more irregular character of the fever, the absence of rose spots and of splenic enlargement, and the presence of leucocytosis. Herpes labialis is very uncommon in typhoid. Blood-examination for the presence of *B. typhosus* and Widal's agglutination reaction are likely to be positive in the case of typhoid. The prolonged stage already referred to, in which residual abscesses appear, has sometimes been mistaken for tuberculous peritonitis. A careful bacteriological examination of the pus will clear up the nature of such cases.

The *circumscribed* type may closely resemble appendicitis, as already remarked. In both, rectal examination may reveal a tenderness together with a boggy sensation to the examining finger on the right side of the pelvis.

Prognosis.—The prognosis in the *circumscribed* variety (which accounts for about 50 per cent. of all cases) is fairly good, the mortality being about 15 per cent. In the *diffuse* form recovery is rare, the mortality being as high as 90 per cent. This high mortality is explained by the fact that surgery is powerless to deal with the septicæmia which is the underlying cause.

Treatment. *Circumscribed form.*—Though spontaneous recovery is not impossible, prompt incision and drainage afford the best chance of saving life. The character of the exudate renders thorough drainage of the peritoneum difficult. The fluid may be removed by using gauze plugs, but injury to the peritoneum from too much manipulation must be avoided. Some pediatricists are opposed to operation except where a residual abscess exists, but as it is not possible to determine without doubt what the cause of the peritonitis is in every case, and as so many cases of peritoneal inflammation prove

fatal if left, the wiser course is the one we have indicated.

The value of intraspinal anæsthesia, especially if respiratory embarrassment exist, should be borne in mind.

In the *diffuse* form of peritonitis surgical interference should be advocated unless other evident lesions coexist, such as pneumonia or meningitis.

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PERITONITIS, TUBERCULOUS.—Tuberculous peritonitis is very frequently associated with the presence of tubercle in some other part of the body. Thus, it may be secondary to tuberculous ulceration of the intestine, to caseating mesenteric glands, to tuberculous disease of the Fallopian tubes in women or to tuberculous infection of the epididymis or vesiculæ seminales in men. In other cases it is secondary to pulmonary involvement, and in yet others the peritoneum may be affected simultaneously with other serous membranes such as the pleura and pericardium (polyserositis, q.v.). In some instances tuberculous peritonitis follows contusion of the abdomen. The organisms may reach the peritoneum by way of the blood or by the lymph-stream, or they may be poured directly into the cavity from an exposed caseating focus, e.g. mesenteric gland, Fallopian tube. In the latter event the bacilli become more or less generally disseminated by the peristaltic movements of the intestines.

Various forms of tuberculous peritonitis are recognized, their diversity depending upon variations in the virulence of the invading organism and in the resistance of the patient.

1. **Acute milary tuberculosis of the peritoneum.**—The symptoms may be similar to those occurring in cases of acute peritonitis due to pyogenic organisms. The exudate may be sero-fibrinous or hæmorrhagic. The peritoneal infection is frequently part of a generalized milary tuberculosis in which other serous membranes and viscera are involved. In these cases the peritoneum becomes infected by way of the blood-stream, and the prominence of the meningeal or constitutional symptoms may mask the signs of peritoneal involvement. At autopsy minute grey tubercles are seen in or beneath the peritoneum, and death may take place before caseation occurs.

2. **Caseating or ulcerative tuberculous peritonitis.**—In this variety neighbouring groups of tubercles become confluent and undergo caseation and liquefaction. Thus the interstices

between adjacent coils of intestine become filled with turbid fluid. These little collections become encysted by the formation of granulation tissue which springs from and binds together neighbouring inflamed coils. Rupture of the tuberculous abscess into the bowel may occur, and, should intestinal contents escape, a localized faecal abscess results. In the event of erosion of the abdominal wall taking place a *faecal fistula* will be produced. Faecal fistulae are most frequently seen at the umbilicus. Should one of these sacculated collections of fluid burst into two adjacent coils of intestine the condition known as a *fistula bimucosa* is established. It is sometimes possible to recognize clinically the existence of such a short circuit. Suppose, for instance, a fistulous communication between the upper part of the jejunum and the descending colon to have developed, not only will the patient pass partially digested food in the stools, but wasting of greater rapidity than the original disease accounts for will result. In less severe infections it may happen that while caseation is going on in some regions of the abdomen, fibrosis is taking place in others. Thus the omentum not infrequently shows thickening and contraction, and forms a more or less horizontal roll across the upper part of the abdomen.

3. Ascitic form.—In this variety an excessive amount of fluid collects in the peritoneal cavity, and the abdomen may show as great an enlargement as in cases of ascites secondary to cirrhosis of the liver. In some of these cases alternating dullness may be absent. The infiltrated mesentery becomes shortened to such an extent that it no longer permits the gas-containing intestine to float upon the surface of the fluid when the patient is turned on to his side. Laparotomy is frequently successful in the treatment of this variety. It would appear that the sudden fall of intra-abdominal pressure brought about by the escape of the ascitic fluid, the bactericidal power of which has been exhausted, leads to a rapid transudation of blood-plasma rich in specific antibodies. This, coming into contact with the infecting organisms, leads to their destruction and a gradual termination of the disease.

4. Fibroid tuberculosis of the peritoneum.—Here the patient's resistance to tuberculous infection is moderately high. The tuberculous granulations, instead of undergoing caseation, progress to the formation of fibrous tissue. With the lapse of time the fibrous adhesions

are so dense and the mesentery so much shrunken that the lumen of the bowel becomes narrowed and distorted, and finally symptoms of intestinal obstruction show themselves.

Symptomatology.—Tuberculous peritonitis is notoriously variable in its clinical manifestations. In some cases the disease is latent, and a tuberculous infection of the peritoneum is only discovered at operation or autopsy. Except when constituting a terminal infection in some chronic visceral disease, like cirrhosis of the liver, tuberculous peritonitis is rarely met with after middle life. It is not uncommon in childhood. In the more acute forms the disease may simulate appendicitis or intestinal obstruction; when less acute, typhoid fever may have to be excluded. In the more chronic forms of the disease, such as are more commonly seen, the onset is insidious and the affected person refers in his history to a few weeks of indefinite malaise, anorexia, and an increasing sense of fatigue. He begins to suffer from abdominal pain, may notice that his abdomen is becoming distended, and finds that he is losing weight. There may be troublesome irregularity of the bowels, in some cases diarrhoea—when intestinal ulceration is present—in others constipation. Under observation, irregular variations of temperature soon become manifest, though it is by no means rare to find the temperature subnormal.

The physical signs vary according to the type present, but in the commoner fibro-caseous form the abdomen is found to be moderately distended and a peculiar “doughy” sensation may be imparted to the hand. In some cases the “rolled-up” omentum can be easily identified, and there may be indefinite masses in other parts of the abdomen. These are due to portions of matted intestine, in some cases enclosing liquefied caseous material, to coils irregularly distended by gas, and less commonly to enlarged glands. In the fibroid form of the disease they may also be due to faecal accumulations.

As the case proceeds the abdomen becomes more “lumpy,” and its increasing enlargement contrasts strikingly with the wasting of the limbs and the thorax. The umbilicus becomes prominent and red. There are night-sweats, diarrhoea increases in frequency, and wasting is progressive. The skin is shrunken and often pigmented, the anaemia more pronounced, and the patient slowly sinks from exhaustion, hastened possibly by the formation of a faecal fistula. In other cases a perforative peritonitis,

PERITONITIS, TUBERCULOUS

intestinal obstruction, or generalized tuberculosis brings life to an end.

Diagnosis.—In early stages this disorder may be difficult to distinguish from any slow wasting disease, but the presence of abdominal pain in a patient under middle age who is slowly losing flesh, should always suggest the possibility of tuberculous peritonitis. Blood-culture, a positive agglutination reaction, and leucopenia will usually serve to distinguish *typhoid fever* from acute cases of tuberculous peritonitis. *Malignant disease of the peritoneum* may resemble tuberculous peritonitis. In both a blood-stained effusion may occur. Malignant disease would be unlikely in a child. Fever is more in favour of the tuberculous lesion, whereas a rapidly developing cachexia, the occurrence of nodules round about the umbilicus, and the presence of enlarged inguinal glands are more suggestive of malignancy. *Cystic tumours of the ovary* sometimes give rise to confusion. Vaginal examination may afford a means of elucidation. *Addison's disease* may be suggested when diffuse pigmentation of the skin occurs. In cases in which ascites is present, if it can be made out that the fluid is encysted, or if the "rolled-up" omentum can be palpated, a diagnosis of tuberculous peritonitis is likely to be correct. An encysted *pneumococcal peritonitis* may be impossible to distinguish without a bacteriological examination of the ascitic fluid, nor must it be forgotten that a secondary invasion by pyogenic organisms is not a very rare event in tuberculous peritoneal effusions.

Prognosis.—The more acute the symptoms the worse is the prognosis. The outlook in ascitic cases is favourable, less so in the fibrous variety, while in the caseous type recovery is much less likely, and it has been well said that a faecal fistula is as good as a death-warrant. In fatal cases death usually occurs from six months to two years from the date of onset; patients surviving beyond this time are likely to recover, and it is remarkable how a badly matted abdomen may eventually get well. Infants under two usually succumb to generalized tuberculosis.

Treatment.—The general treatment will be similar to that for tuberculosis in any other part of the body. The patient should enjoy as much fresh air day and night as possible. Sunlight is an advantage. He should remain in bed as long as fever is present. The diet should be abundant, and should contain plenty of fat in the form of cream, butter, fat bacon,

etc. In diarrhoeal cases a milk diet is indicated and arrowroot from time to time is useful. If the milk is not obtained from tuberculin-tested cows it should be boiled before being given. Locally, inunction of equal parts of ung. hydrargyri oleatis and of vaselin may be performed once a day for several weeks; $\frac{1}{4}$ –1 dr. of the mixture may be used for an adult; should diarrhoea occur it is usual to discontinue the inunction for a time. Internally, cod-liver oil and malt, creosote, or syr. ferri iodidi may be employed. In cases in which diarrhoea is frequent, Dover's powder 2–5 gr., combined with tannalbin 10 gr., and given in cachet, is of service.

Injections of tuberculin often lead to improvement. In ascitic cases $\frac{1}{100000}$ mg. of T.R. may be given as an initial dose and gradually increased till the temperature becomes normal. The injections are to be given every four or five days, and treatment usually lasts for four, five or six weeks. When convalescence is attained it is usual to continue the injections once a week or fortnight for six months. Tuberculin is less successful in the caseous form of the disease. The initial dose should be about $\frac{1}{100000}$ mg. of T.R., increased very cautiously. A rise of temperature or increase of pain is taken as an indication for temporarily stopping the treatment.

Ascitic cases not subsiding under five or six weeks of medical treatment should be treated by laparotomy. This allows of the escape of the accumulated fluid, and rapid improvement frequently results. The wound is closed without drainage. Exceptionally, a second laparotomy is necessary, and in these cases a limited exploration may reveal the presence of a caseating gland which may wisely be removed.

Laparotomy is not advisable in other varieties unless to relieve obstruction or to deal with a perforation. It is never wise to incise the prominent umbilicus; in many cases a tuberculous abscess lies beneath, and sooner or later a faecal fistula is likely to form. It is not expedient to attempt to treat faecal fistulae by surgical measures; in such cases extensive peritoneal adhesions are often present, and the walls of the intestine are too soft and friable to permit of manipulation.

C. E. LAKIN.

PERITONSILLITIS (see QUINSY).

PERIURETHRAL ABSCESS (see ABSCESS, PERIURETHRAL).

PHARYNGITIS, ACUTE

PERLÈOHE (see IMPETIGO CONTAGIOSA).

PERNICIOUS ANÆMIA (see ANÆMIA).

PERNICIOUS VOMITING OF PREGNANCY (see VOMITING, PERNICIOUS, OF PREGNANCY).

PERONEAL MUSCULAR ATROPHY (see MUSCULAR ATROPHY, PERONEAL).

PERTHES' DISEASE (see PSEUDO-COX-ALGIA).

PERTUSSIS (see WHOOPING-COUGH).

PES OAVUS (see TALIPES).

PETIT MAL (see EPILEPSY).

PHARYNGITIS, ACUTE.—An acute inflammation of the mucous membrane of the pharynx, resulting in congestion, and in severe attacks leading to infiltration with excessive secretion.

Etiology.—The causes are :

1. Predisposing.

- (a) Lowered vitality—the result of under-feeding, living in vitiated, smoky or dusty atmospheres.
- (b) Constitutional conditions, such as gout, rheumatism, disorders of the digestive tract.
- (c) Catarrhal and septic conditions of the nose and nasal accessory sinuses, naso-pharynx, and larynx.
- (d) Excessive indulgence in alcohol and tobacco.

2. Exciting.

- (a) Traumatism, such as hot or corrosive fluids; after wounds caused by foreign bodies, or after operations in the nose or naso-pharyngeal area, when much sponging has been done.
- (b) Inflammatory conditions of the nose, accessory sinuses, larynx, or naso-pharynx.
- (c) Many of the infectious fevers.

Pathology.—There is considerable inflammation, with congestion and swelling of the mucous surfaces and free secretion. Later this is followed by a small-celled infiltration of the connective tissue, the uvula becoming enlarged and oedematous, perhaps to an enormous degree. In severe septic cases (which are usually streptococcic), sloughs form on the posterior wall, and the tonsils and uvula may become gangrenous. There is a

marked tendency for the disease to spread down to the epiglottis and larynx.

Symptoms.—In the simpler catarrhal type there is an early sense of pain and discomfort in the throat, especially on swallowing, with a constant hawking as if due to a foreign body; the temperature is slightly raised to 100-101° F. The glands in the neck may become enlarged and tender, and if there is much oedema of the uvula the voice alters. In the more severe infectious types the temperature runs up to 103-104° F., initiated by a rigor, with great pain, especially on deglutition. The general condition is one of septic poisoning, and may progress to delirium and coma. The glands of the neck become much swollen, and abscess-formation may follow.

Prognosis.—The outlook is good in the simple types, recovery taking place in from two to fourteen days. In the severe types there may be serious danger to life as the result of the septic poisoning.

Treatment.—In the acute catarrhal type the patient should be put to bed and a dose of calomel (3-5 gr.) given at night, with a saline in the morning; all alcohol and smoking must be forbidden. If there is much pain, salicylate of soda in doses of 5-10 gr. can be given every two hours until it is relieved. The throat and nose should be sprayed with an alkaline lotion such as—

Ry Sod. bicarb. } aa ʒi.
Sod. biber. }
Sod. salicyl. gr. iiii.
Menthol } aa gr. i.
Thymol }
Glycer. ʒi.
Aq. ʒiv.

One part to 10 parts of warm water in a spray, to be followed by a spray in an atomizer :

Ry Menthol gr. xx.
Thymol gr. x.
Eucalyptol ℥xx.
Ol. cubeb. ℥xxx.
Paroleine ad ʒiv.

A useful lozenge is :

Ry Menthol gr. ʒi.
Ol. eucalyp. ℥i.

M. ft. trochis. One to be sucked every 2-3 hours.

No attempt should be made at painting in the acute stage. Later the following gives excellent results :—

Ry Menthol } aa ʒss.
Guaiaicol }
Ol. amygd. dulc. ʒi.
M. ft. pigment.

PHARYNGITIS, CHRONIC

In the severe infective type the case must first be treated with a calomel purgative, and antistreptococcic serum administered in doses of 10–20 c.c. Internally, quinine in 5-gr. doses three times daily, and perchloride of iron in 20–30-min. doses every four hours, should be given. Any signs of heart failure must be combated by hypodermic administration of adrenalin chloride 1 in 1,000, in 5-min. doses, four-hourly, or strychnine or digitalin; and alcohol must also be administered. Locally, if there is much muco-pus or slough, hydrogen peroxide in a spray, followed by the oily solution mentioned above, is the best. Steaming the pharynx with the following is soothing and efficacious:—

℞ Menthol gr. xl.
Sp. rectif. ʒi.
One drachm to a pint of boiling water.

J. GAY FRENCH.

PHARYNGITIS, CHRONIC.—The following forms are distinguished, viz. Simple, Hypertrophic, and Atrophic.

SIMPLE AND HYPERTROPHIC PHARYNGITIS.

The causes of these forms of pharyngitis are:

(1) Frequent attacks of acute pharyngitis. (2) Digestive troubles, especially in gouty or rheumatic subjects. (3) Disease of the nose and nasal accessory sinuses. (4) Working in dusty or vitiated atmospheres. (5) Misuse of the voice. (6) Excesses in alcohol or tobacco.

Pathology.—The mucous membrane becomes congested and of a dark-reddish colour; the blood-vessels pass into a state of chronic dilatation, and the lymphoid nodules surrounding the openings of the mucous glands hypertrophy. At the same time the lymphoid masses on each side, known as the salpingo-pharyngeal folds, increase and appear as two inflamed strands behind the posterior pillars of the fauces, one on each side, a condition to which the name of *lateral pharyngitis* has been given.

Symptoms.—There is usually a dryness and soreness at the back of the throat, accompanied by frequent attacks of violent hawking. The patient often feels as though a foreign body such as a hair were present. The voice is complained of as being weak and tiring easily.

Treatment.—A careful examination should be made as to the cause—errors of digestion or intranasal trouble, for instance—and this

should receive adequate treatment. Alcohol and tobacco should be prohibited; a misused voice should receive rest, followed by a proper course of voice training. In young children removal of tonsils and adenoids will often lead to a cure. Locally, an alkaline spray should be used both into the pharynx and through the nostrils. The oily solution in an atomizer mentioned under PHARYNGITIS, ACUTE, is very useful, as is also the menthol and guaiacol paint which follows it. If there are a number of enlarged follicles they can be destroyed by a galvano-cautery point under cocaine, but not more than four should be dealt with at one sitting. The cautery can also be applied effectually to the inflamed lateral bands. It is important to remember that the cautery should be used for the pharynx as little as possible, lest an atrophic condition be originated. A solution of Mandl's paint, or a 10-per-cent. solution of silver nitrate, is also useful.

CHRONIC ATROPHIC PHARYNGITIS.—This form of pharyngitis usually occurs in cases where there is an atrophic condition in the nose, or as the result of prolonged nasal sepsis.

Pathology.—There is a gradual atrophy of the mucous glands with obliteration of the blood-vessels.

Symptoms.—Great dryness and irritation in the pharynx are commonly complained of. There may be considerable hawking and coughing as the result of the formation of dry crusts, and hoarseness and cough may be present as the condition advances and involves the larynx.

Prognosis.—In the early stage the prospect is good; later, though the changes in the mucosa are permanent, as a rule the process can be arrested and relief given.

Treatment must first be directed to the primary cause, which is almost invariably found in the nose. The pharynx should be sprayed with an alkaline lotion, or, if the crusts are very adherent, with a 5-volume solution of hydrogen peroxide. This should be followed by the application of either Mandl's solution or:

℞ Ichthyol aa ʒii.
Glycer. i
Aq. ad ʒi.

General treatment must be given to combat the anæmia and the poor state of health often present in these cases. J. GAY FRENCH.

PHARYNX, NEW GROWTHS OF

PHARYNX, NEW GROWTHS OF.—The following **innocent growths** are met with :—

1. Papilloma.
2. Fibroma.
3. Angioma.
4. Adenoma.
5. Dermoid cyst.
6. Endothelioma.

1. **Papilloma** is the commonest of the innocent growths, and is usually seen on the uvula, the edges of the palate, or the anterior pillars of the fauces. It is soft, and generally pedunculated. It rarely grows to a large size, and is of a pale-pink colour with a cauliflower surface. It seldom gives rise to symptoms, but when growing to a fair size may cause discomfort in the throat with a tickling sensation and sometimes cough.

Treatment.—A papilloma is usually not interfered with unless it is growing large and causing symptoms, when it can be removed under cocaine anæsthesia. The papilloma is seized with a toothed forceps and pulled forward, the base being divided either with scissors or with a snare. This ensures removal of the whole growth.

2. **Fibroma** is rare. It occurs on the tonsils or on the arches of the palate. In appearance it is a round, pink, sessile growth, hard to the touch, and it is usually free from symptoms. It is dealt with in the same way as a papilloma, except that a general anæsthetic may be necessary.

3. **Angioma** is extremely rare. It may be found either at the junction of the hard and soft palates, or on the posterior wall of the pharynx. It may give rise to discomfort, pain, and dysphagia.

Treatment.—An angioma should always be removed, and a general anæsthetic is necessary. The incision should not approach the edges of the growth, which can be dissected away with a blunt dissector. Hæmorrhage from any big vessel is, as a rule, easily controlled by means of pressure-forceps, and, should there be general oozing, a Paquelin's cautery used at a dull-red heat proves satisfactory.

4. **Adenoma** occurs in the palate, and may grow to a large size. If large, it may interfere with swallowing and speech, and give rise to pain.

Treatment.—Remove under a general anæsthetic. An incision is usually made over the growth, which can then generally be easily shelled out, and the incision closed with a few stitches.

5. **Dermoid cyst** is extremely rare. It is

situated in the middle line—probably connected with the foramen cæcum. The tumour is pedunculated, and can be easily removed with a snare.

6. **Endothelioma.**—The usual position is in the tonsil, soft palate, or posterior pharyngeal wall. The growth is smooth, round, pink and firm. The symptoms vary with the position: usually dysphagia, alteration in speech, and pain and discomfort are complained of. Dyspnoea may occur if the growth is obstructing the respiratory passage. The growth has a definite capsule.

Treatment.—Complete removal. This is done by an incision over the growth, which can then easily be shelled out.

Malignant growths.—Carcinoma and sarcoma occur fairly frequently in the pharynx, both as primary growths and as metastases. As the symptoms which they provoke are similar, both types will be considered together.

These neoplasms may occur in any part of the pharynx. Primary sarcoma usually runs a rapid and fatal course, though numerous cases are recorded in which it has continued for six to eight years before terminating fatally. The cervical glands enlarge early, and ulceration takes place rapidly in the primary growth.

Carcinoma rarely occurs before the age of 40, and is commoner in males. The usual type is the squamous-celled.

Symptoms largely depend upon the position. The earliest is a sense of discomfort and fullness in the throat; pain is frequent, especially in carcinoma. The cervical glands are early implicated. If the growth is in the respiratory tract, dyspnoea becomes pronounced. Later, severe pain, dysphagia, odynphagia, dyspnoea, foetid breath and excessive salivation, with rapid wasting, all become evident.

Diagnosis.—In the early stages the chief difficulty concerns tertiary syphilis. The administration of potassium iodide and the Wassermann reaction should determine the question, and a removal and microscopical examination of a portion of the growth under cocaine is decisive.

Prognosis and treatment.—Unless the growth is removed early it proves fatal. Unfortunately, even in this stage removal is often followed by recurrence. In the later stages palliative treatment must be had recourse to. Radium and diathermy are always worth trying. The former in sarcoma of the nasopharynx has recently proved of great benefit in a number of cases. J. GAY FRENCH.

PHARYNX, SPASM OF.—Pharyngeal spasm may be purely functional, of which the best example is the *globus hystericus*; or it may be due to disease of the central nervous system, as in *tabes dorsalis*, cerebral tumours, general paralysis and meningitis. The condition is also seen in tetanus and in hydrophobia. The commonest type is the functional; in this, rhythmic movements of the pharyngeal walls may be seen, possibly accompanied by an audible click. It is usually met with in women. One variety occurs in young children, and may lead to difficulty in swallowing.

Treatment.—In the organic type little can be done, but in the functional, change of air and the administration of bromides with valerian give the best results. Faradism and the high-frequency current to the neck have both been recommended. J. GAY FRENCH.

PHARYNX, SYPHILIS OF.—In the acquired form syphilis is met with in the pharynx in the primary, secondary and tertiary stages. In the congenital variety, secondary and tertiary stages are recognized.

ACQUIRED SYPHILIS

PRIMARY.—The chancre is usually seen on the tonsil, palate, faucial pillars, or on the posterior wall of the pharynx. The infection is transmitted by kissing, perverted sexuality, or through infected implements such as knives, forks, spoons, etc.

Symptoms.—As a rule, there is a sore throat, which is very pronounced, with some pain on deglutition.

Pathology.—The affected portion is swollen and hard to the touch, its centre being ulcerated and covered with a greyish sloughy membrane. There is early enlargement of the submaxillary glands, which are hard, without fixation, and have no tendency to suppurate or involve the skin.

Diagnosis depends on the appearance of the lesion, the characteristic swelling of the glands, the detection of the *Spironema pallidum*, and the establishment of a positive Wassermann reaction. It may be necessary to wait for the appearance of the secondary lesions.

Prognosis is good with early treatment.

Treatment.—The local treatment consists in keeping the patient's mouth and teeth scrupulously clean, and directing him to gargle with a solution of *lotio nigra* $\frac{1}{2}$ oz., potass. chlorate 15 gr., water to 1 oz. The general treatment is the more important. (See SYPHILIS.)

SECONDARY.—Two main forms are seen, erythema and the mucous patch. *Erythema* is the commoner; in it the mucous membrane is thickened and of a purple-red colour. It is usually situated on the palate and tonsils, and frequently takes the shape of two crescentic patches spreading from the anterior pillars to the uvula. The *mucous patch* is situated, in order of frequency on the tonsil, uvula, soft palate, pillars of the fauces and, rarely, the posterior pharyngeal wall. These patches are rounded and slightly raised, and covered with a bluish-white membrane, the surrounding area being considerably inflamed.

Symptoms.—Sore throat with some pain on swallowing is complained of.

Prognosis and treatment.—If treated early, the mucous patch gradually disappears. The gargle of black wash and potassium chlorate described above is efficacious. Mucous patches should be dried and touched with a 25 per cent. solution of silver nitrate. Alcohol and tobacco must be strictly forbidden. Generally, antisyphilitic treatment must, of course, be adopted.

TERTIARY.—In the pharynx the tertiary lesions met with are (1) gummata, (2) ulceration, (3) scars and adhesions.

(1) *Gummata* may be diffuse or circumscribed, and are usually seen on the posterior pharyngeal wall, palate, faucial pillars, or tonsils. The swellings are rounded, hard, and grow rapidly. Those which are of the circumscribed type show a tendency to ulcerate early, but those which are diffuse are more persistent and less prone to ulceration. Pain, especially on deglutition, and alteration of the voice are the chief symptoms complained of.

(2) Tertiary *ulcerations* result from the breaking down of gummata, and are of two varieties, (1) the serpinginous superficial, and (2) the deep crateriform. The former are seen in the early tertiary period, the latter in the later stages.

(3) *Scars and adhesions.*—After healing has been established, there is a marked tendency to the formation of cicatrices. Extensive scars may be seen on the post-pharyngeal wall, and deformities occur from the resulting adhesions. Thus the naso-pharyngeal passage may be completely occluded, or very much narrowed, by adhesions between the soft palate and the posterior wall of the pharynx, or severe dysphagia be brought about by adhesions between the posterior pharyngeal wall and the base of the tongue.

PHARYNX, TUBERCULOSIS OF

Diagnosis is arrived at by obtaining a history of syphilis and by the local appearances. A positive Wassermann reaction and successful results by treatment with mercury and iodides are confirmatory.

Treatment.—Of chief importance is the general treatment (*see* SYPHILIS). Local treatment is also essential, and consists in keeping the mouth and teeth scrupulously clean. Lotion nigra makes an excellent mouth-wash and gargle in these conditions. The ulcerations should be touched with a 25- to 50-per-cent. solution of silver nitrate, or fused silver nitrate on a probe. Adhesions may be treated by stretching or removal, but it must always be borne in mind that they show a tendency towards recurrence, and where possible they should be left alone. Large perforations through the hard palate are best dealt with by adjusting an obturator.

CONGENITAL SYPHILIS

In congenital syphilis of the pharynx the secondary and tertiary manifestations are similar to those seen in the acquired type. The secondary lesions frequently occur within the first six months of life, taking the form of an erythema or a mucous patch; the tertiary are most frequently seen at puberty, usually as a gummatous infiltration or ulceration, and are in every way similar to the acquired varieties.

J. GAY FRENCH.

PHARYNX, TUBERCULOSIS OF,

ACUTE.—Acute tuberculosis of the pharynx occurs as the miliary form and is almost invariably secondary to tuberculosis of the lungs. Its appearance is of serious omen, the cases almost invariably terminating fatally. It is seen in the mucous membrane of the soft palate, uvula, faucial arches, and, rarely, in the tonsils and posterior pharyngeal wall. The tubercles at first present the appearance of pink, discrete nodules; these rapidly coalesce and break down into shallow ulcers, irregular in shape, with undermined edges, sloughy base and a contiguous reddish areola, the surrounding mucous membrane being extremely anæmic. The tissues around the ulcers are usually œdematous, and the cervical glands become enlarged.

Symptoms.—Severe pain is experienced, and is increased on any attempt at swallowing. The patient is seriously ill with pulmonary or general tuberculosis, the temperature is consequently raised, and there is rapid wasting.

Diagnosis.—The general condition of the patient, the local appearance in the pharynx and the presence of cough and expectoration are the chief points in diagnosis. Examination of the sputum reveals tubercle bacilli.

Treatment.—The treatment should chiefly be directed to the antecedent tuberculosis of the lungs. Locally, palliative treatment only can be adopted, for the patient's condition is too serious to allow of energetic measures. The pharynx should be sprayed with an alkaline lotion such as Dobell's, and insufflated with a powder to allay the pain and permit food to be swallowed in comfort. The best powder is a mixture containing equal parts of orthoform, anæsthesin and iodoform; it can be insufflated with advantage half an hour before the administration of food.

J. GAY FRENCH.

PHARYNX, TUBERCULOSIS OF, CHRONIC.

—This condition is seen as lupus, affecting the soft palate, the faucial arches, and the posterior pharyngeal wall, and presenting itself as minute, discrete, pinkish nodules with the typical apple-jelly centres. The whole area appears thickened, and breaks down into superficial ulcerations which show the characteristic tendency to heal at one edge while spreading at another. Considerable destruction may take place, and healing may leave pronounced deformity as the result of cicatrization. The healed parts often show a tendency to break down easily.

Symptoms.—The symptoms are usually mild. There may be some thickening of the voice, and discomfort, with occasionally a tendency to regurgitate liquid through the nose should the palate be extensively diseased.

Prognosis.—The disease is slow but progressive, and unless arrested leads to extensive destruction of the tissues. It can, however, be arrested by treatment.

Diagnosis.—*Syphilis* provides the chief difficulty in diagnosis. Syphilis, however, is much more rapid, responds to antisyphilitic treatment, and reacts positively to Wassermann's test. Removal of a small portion for microscopical examination reveals the nature of the disease.

Treatment.—Fresh air and good food with cod-liver oil and malt are indicated. Of drugs, arsenic is the most satisfactory, and should be given three times daily, commencing with 3-min. doses and increasing up to 10-min.

PHIMOSIS

The application of the electric cautery to the nodules is useful. If there are sluggish ulcers, they should be cleansed, cocaineized, and receive an application of fused silver nitrate on a probe. Curetting is rarely necessary.

J. GAY FRENCH.

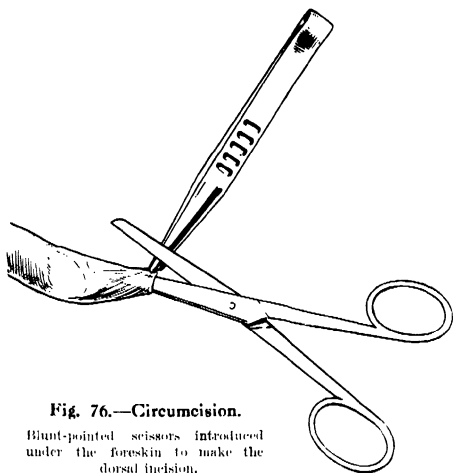
PHIMOSIS.—A condition in which the preputial orifice is not large enough to allow of the retraction of the foreskin over the glans. It may be either congenital or acquired. The acquired form may be inflammatory and due to the oedema of the prepuce sometimes associated with gonorrhoea, venereal sores, or non-venereal balanitis, or it may be cicatricial. In the congenital variety there are usually firm adhesions of the mucous membrane of the prepuce to the surface of the glans. If the orifice of the prepuce is pinpoint in size there may be an obstruction to micturition, and the foreskin will balloon out at each evacuation of the bladder. Certain reflex phenomena have been associated with phimosis and its accompanying adhesions—retention of urine, nocturnal enuresis, convulsions, night-terrors, colic, indigestion. Exceptionally, phimosis is the cause of repeated attacks of balanitis and the formation of preputial concretions. Although congenital phimosis may disappear with growth as the child reaches seven or eight years, it is better, when the orifice is very small, to recommend and perform circumcision.

Treatment.—When there is inflammatory phimosis the foreskin should not be removed immediately. A simple incision made along the dorsum will give drainage and allow local treatment to be applied. When the septic process has disappeared, circumcision may be done should the patient desire it.

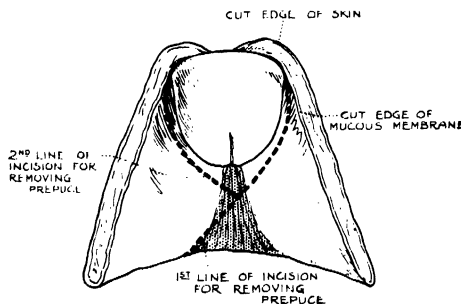
In performing **circumcision** it is best to employ general anaesthesia. It is important to remove the proper amount of skin and mucous membrane. Without dragging the foreskin forwards, make four nicks with the knife or four snips with scissors to mark where the skin should be cut through. Three of these marks—one on the dorsum and one each side—should be placed at a level just distal to the corona of the glans felt through the foreskin. The fourth nick is made on the under-surface just where the frænum can be felt to be attached to the glans.

There are two methods of removing the prepuce: (1) It is split up the dorsum with blunt-pointed scissors until the dorsal mark has been reached (Fig. 76). To make room for the blade

of the scissors a probe must first be insinuated between the glans and foreskin along this line to separate adhesions. The dorsal cut



being completed, the adherent foreskin must be peeled off the glans until the groove behind the corona is exposed, setting free the retained smegma. Both skin and mucous membrane are then cut off along the line already marked out on the skin. More mucous membrane must be trimmed away so that only a narrow strip a little more than $\frac{1}{8}$ in. wide is left. (Fig. 77.)



(2) The foreskin is dragged forwards and pulled through the slit in a flat piece of metal to protect the glans from injury, or it may be

PHIMOSIS

grasped between the handles of a pair of sinus forceps which function similarly. The marks on the foreskin should be just beyond the metal plate or forceps, the tissue distal to which is cut off flush with a scalpel. The skin retracts. The mucous membrane is then split up the dorsum, peeled back, and trimmed off as in the first method.

The frænar artery, which runs backwards from glans to prepuce, is tied with fine catgut (Fig. 78). The other bleeding vessels are twisted in infants, ligated in adults. The suturing is done with fine non-chromicized catgut No. 00, and is begun at the frænum. The needle should always pass from mucous membrane to skin.

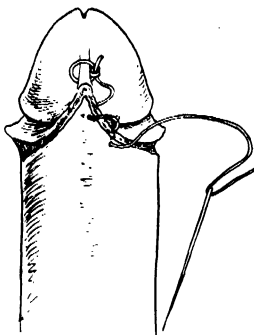


Fig. 78.—Circumcision.

To show how the artery to the frænum is tied, and the end of the suture used to stitch mucosa to the skin.

A continuous suture, if it be not pulled too tight, may be used, or interrupted sutures. The meatus must always be carefully inspected. If it is pinpoint, meatotomy downwards should be done, and care taken in the after-treatment to prevent it from contracting up again. The dressing should be sterilized vaselin spread on narrow ribbon gauze, or ung. hydrarg. oxid. flav. used similarly. A pad of wool with a T-bandage is placed over this. A good dressing for small infants is boric-acid albumen, made by stirring thoroughly a drachm of boric-acid powder into the white of a perfectly fresh egg. With a camel-hair brush this is painted over the line of suture at the end of the operation. No other dressing is used, the ordinary napkin being put on. Every time the napkin is changed the operation area is again painted. There is no need to remove the catgut sutures.

C. A. PANNETT.

PHLEBITIS

PHLEBITIS.—Inflammation of the walls of a vein. Since such inflammation almost always leads to clotting of the blood inside the vein (venous thrombosis), the two conditions cannot well be separated.

Etiology.—The causes are :

1. Traumatism. Severe or even slight injury may so damage the vein as to induce phlebitis and thrombosis.

2. Infection, whether spreading from neighbouring parts or carried to the vein by the blood-stream.

3. Gout.

4. Following primary thrombosis in such conditions as anæmia, cancerous cachexia, and convalescence from fevers. It is possible that in some of these states there may be toxins which directly cause phlebitis.

5. Post-operative, due to traumatism or infection.

Symptomatology.—Pain is felt along the course of the affected vein, which is tender on pressure. Swelling and cedema are noticed, both locally and in the parts distal to the inflamed vein. If the inflammation is septic, suppuration may ensue, and should the clot in the vein be disintegrated, particles may be carried into the general circulation and cause pyæmia. In non-infective cases there is slight fever and increase of pulse-rate; in infective cases the temperature may be very irregular.

Special varieties. *Veins of leg.*—Superficial varicose veins of the leg frequently become inflamed owing to slight injury, or to infection from small ulcers. A firm cord can often be seen and felt under the skin, but in a fat subject the internal saphenous vein cannot always easily be palpated. Phlebitis of the deep veins of the leg is not uncommon, especially in gouty subjects; painful swelling of the calf and ankle region and a limp on trying to walk may be the only symptoms. Phlebitis and thrombosis of the femoral vein cause great cedema of the lower limb.

Lateral sinus.—Infection of the lateral sinus is a serious complication of otitis media. It is accompanied by irregular fever with occasional rigors, tenderness along the course of the jugular vein, and the general symptoms of pyæmia. (See LATERAL-SINUS THROMBOSIS.)

Cavernous-sinus infection causes symptoms of pyæmia with local swelling and congestion of the conjunctiva and some protrusion of the corresponding eyeball. (See CEREBRAL SINUSES, THROMBOSIS OF.)

Portal vein.—Phlebitis of the veins of the

portal system (pylephlebitis) is a serious condition usually following on an infective focus in the intestinal tract, e.g. appendicitis. Irregular fever, rigors, tenderness over the liver, general symptoms of toxæmia, and occasionally jaundice and hæmatemesis point to the condition.

The *pampiniform plexus* of veins may become inflamed and form a tender swelling reaching from the testicle to the abdominal cavity. If suppuration occurs, the ensuing abscess may extend into the retroperitoneal abdominal tissues.

The *inferior vena cava* may thrombose, but whether owing to previous phlebitis or consequent on some condition of the blood is doubtful. If the patient survive, there will be great swelling of the lower extremities, and compensatory enlargement of the superficial veins on the abdomen.

Diagnosis.—Thrombosis of superficial veins is usually easily detected, but if over the tibia may be mistaken for periostitis. Postoperative phlebitis of the saphenous or femoral vein (especially on the left side) should always be looked for if the temperature or the pulse-rate rises without obvious cause elsewhere. Local pain in these cases is often slight. Unfortunately, sometimes the first symptom is the dislodgment of a clot, causing pulmonary embolism with symptoms of dyspnoea, lividity, lung consolidation and possibly fatal asphyxia.

Infective phlebitis of the lateral sinus and jugular vein, and of the portal vein, are considered under LATERAL-SINUS THROMBOSIS, PYLEPHLEBITIS, SIMPLE, and PYLEPHLEBITIS, SUPPURATIVE. Phlebitis of deep veins of the leg may be mistaken for *muscular rheumatism* if due care be not taken. Infection of the veins of the spermatic cord causes a swelling likely to be mistaken for inflamed or strangulated *hernia* by a careless observer.

Prognosis.—Recovery is the rule in a simple case, but there is always the grave risk of pulmonary embolism urging the practitioner to treat the condition as serious. In septic cases the risk of pyæmia makes the prognosis more serious. In sinus phlebitis death is common.

Treatment. **Non-infective cases.**—Rest in bed for from three to six weeks until pain over the course of the vein has disappeared is imperative. If the vein be in the leg or arm, the limb should be wrapped in cotton-wool, supported on splint or on pillows, and kept perfectly still. The patient should be warned against any undue or sudden movement or

exertion, especially straining at stool. Aperients and enemata to avoid constipation are necessary. Fomentations, or glycerin and belladonna, may be applied locally for the pain. *When all local pain has gone*, gentle massage must be started, and gradually increased if no ill results follow. A firm bandage should be worn afterwards to lessen swelling of the limb.

Infective cases.—In the case of superficial veins an incision must be made into any abscess which forms. In lateral-sinus infection the sinus must be opened, the jugular vein tied in the neck, the septic material washed away and drainage instituted. In the leg it is sometimes possible to excise completely a local patch of inflamed and thrombosed veins.

To prevent extension of thrombosis, diminish the coagulability of the blood by giving citric acid or making the patient drink plenty of lemonade. For the treatment of pulmonary embolism, see LUNG, EMBOLISM OF.

ZACHARY COPE.

PHLEBO-ARTERITIS, THROMBOTIC (see THROMBOTIC PHLEBO-ARTERITIS).

PHLEBOTOMUS FEVER (*syn.* Sandfly Fever, Papatasi Fever, Three-day Fever, "Dog Disease").—A benign fever of short duration transmitted by the phlebotomus fly.

Etiology.—New-comers to an area in which the fly is found are very prone to be attacked, perhaps as many as 50 per cent. contracting the disease. This was demonstrated during the recent War, outbreaks occurring among the troops in India, Gallipoli, Mesopotamia, Salonika, Palestine, "Syria," the "Ægean Islands," and Egypt. New arrivals in tropical countries appear to be liable at any time, but in the subtropics the incidence is chiefly in the summer and autumn. The patient's blood is infected, and its injection into men or into monkeys has transmitted the disease. The germ is ultramicroscopic and passes through the finest filters.

The fly (Fig. 79).—Sandflies are minute, delicate, fragile insects which bite by night and against which a mosquito net affords no protection. Their wings are leaf-shaped, and during rest are erect. The legs are long and slender, and wings, body, and antennæ downy. Altogether the insect is about equal in size to a pin's head. Only *P. papatasi* has been shown to transmit the disease, though it is probable that other species do so also. Phlebotomus flies shelter and lay their eggs in

PHLEBOTOMUS FEVER

damp places, cracks in walls and embankments, walls of cellars, heaps of damp stones, cesspools, etc., and may be carried in cargoes from place to place. Their flight is halting and low, and they probably only traverse short distances. The cycle of egg, larva, and imago takes from one month to two months or more to complete, according to the atmospheric conditions.

Symptomatology.—An incubation period of four to seven days is followed by chilliness and malaise, and perhaps a rigor. Giddiness, severe frontal headache, pain at the back of the eyes, and generalized pains in limbs and muscles are complained of. Though drowsy, the patient is irritable and cannot sleep. The face is flushed and puffy, and injection of the

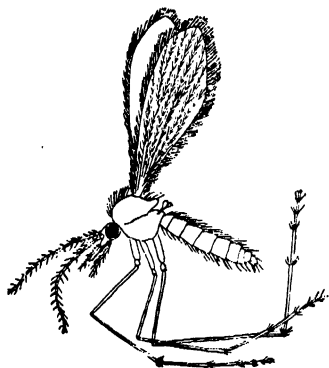


Fig. 79.—The phlebotomus fly (female).
(After Alcock.)

conjunctivæ intense. Vomiting and diarrhoea may occur, but constipation is more usual, accompanied by anorexia and epigastric pain or discomfort. The tongue is coated with a white fur, but clean at the tip and edges. The palate and fauces are injected, and may be studded with small vesicles. The skin is usually dry, but may be clammy. Apart from the congestion of the face, which may extend to the shoulders, there is no rash, though the bites of the fly may produce tender itching papules or even vesicles. Within about twenty-four hours the temperature rises to 101°, 102°, or 104° F., where it remains for another day, and then begins to fall, declining gradually on the third and fourth days. The pulse-rate is little, if at all, increased. The blood shows leucopenia, with a relative decrease in polymorphonuclear cells. Eosinophils are reduced

PHLEGMASIA ALBA DOLENS

during the fever, but increase afterwards. Convalescence may be rapid, but the patient often experiences a period of general weakness and lassitude, depression, sleeplessness, and dyspepsia. Death from this disease is unknown.

Treatment.—Prevention is by avoidance or destruction of the fly, or by treatment of its haunts. Latrines may be fumigated with sulphur, dark rooms and buildings white-washed, damp places drained. Removal to an upper storey in an infected house may out-distance the fly. Repellents are useful; they include camphor and various emollients. Oil of eucalyptus may be rubbed into the skin of exposed parts, or the following ointment, recommended by Balfour:

Ry	Ol. anisi	} aa Miii
	Ol. eucalyp.	
	Ol. tereb.	
	Lanolini ʒi.	

Opium (liq. opii sed. 30 min.) relieves the headache and is the most useful drug.

FREDERICK LANGMEAD.

PHLEBOTOMY (see VENESECTION).

PHLEGMASIA ALBA DOLENS.—"White leg" is the common term for the condition of swelling of the lower limb, with pain and fever, sometimes met with in the puerperium.

Etiology.—The predisposing causes are the increased coagulability of the blood during the puerperium, especially after severe hæmorrhage at delivery, and the comparatively stagnant condition of the venous circulation in the larger veins of the pelvis while the patient is lying in bed. The exciting cause is probably always local pelvic infection. Its virulence may vary greatly, so that its effects may range from a slight thrombotic œdema of the leg to a severe parametritis, of which the phlegmasia dolens is merely one of the manifestations. The infection is usually of the parametric variety, and often follows a deep tear of the cervix.

Pathology.—The essential changes appear to be a thrombo-phlebitis, together with some obstruction of the lymphatics. The existence of the former is easily explained by direct extension of thrombosis from the clot normally existing in the uterine venous sinuses to the veins of the broad ligament and thence to the iliac and femoral veins. The infection, however mild, sets up a phlebitis, which in its turn induces an extension of the thrombosis.

The exact changes in the lymphatics are not so clearly understood, but it is probable

PHLEGMASIA ALBA DOLENS

PIGMENTATION

that a similar process, of extending lymphangitis starting from the broad ligament, causes occlusion of the lymph-channels with resulting lymphatic obstruction in the lower limb. The veins chiefly affected are the iliac and femoral, with frequent extension into the subsidiary deep veins of the limb. The lymphatic glands of the groin are often enlarged, matted together, and very tender. The clot within the vessels is liable to suppurative changes, and thus general sepsis and pyæmia may result.

Symptomatology.—The onset of the disease is usually at the end of the second or the beginning of the third week of the puerperium, in contradistinction to other forms of puerperal fever, which appear within the first week. The chief symptom is severe pain in the leg, generally first felt in the region of the groin or calf, and accompanied by shivering, sudden rise of temperature, and increased pulse-rate. There are headache and malaise. Later, the bowels become confined, and the tongue furred. The pyrexia reaches 101° or 102° F., and, after remaining at that level for a week or ten days, gradually declines. Examination of the leg reveals a uniform swelling of the whole limb, which usually pits on pressure; the skin is tense, white and shiny, and there is considerable tenderness on palpitation, especially in the upper part of the thigh along the courses of the large veins. Sometimes the internal saphenous vein can be felt as a hard, very tender cord. Less commonly the *œdema* is more "solid," and there is no pitting. After a fortnight the general symptoms usually abate, but the limb remains swollen, and only slowly resumes its normal appearance. After all swelling has disappeared, *œdema* of the foot is apt to return when the patient gets up.

Treatment.—The patient should rest completely in bed, with the affected limb immobilized by sandbags and raised on pillows. Movement of the limb, or massage of any description, during the first four weeks is dangerous, inasmuch as a clot may be disturbed. If the pain be severe during the early days, glycerin-and-belladonna fomentations may be applied, and afterwards the limb may be wrapped in cotton-wool. The patient should be kept in bed until there is no further trace of swelling, pitting, or tenderness over the femoral vein. Massage is only permissible at this stage, and is only useful in restoring muscular tone to the limb. Should puerperal sepsis be a predominant feature, suitable treatment for this condition is, of course, necessary.

Citric acid is advised by some writers, on the ground that the blood is thereby rendered less coagulable. It may be given in the form of a lemon daily.

A. W. BOURNE.

PHLYOTENULE (see CONJUNCTIVITIS).

PHOBIAS (see PSYCHASTHENIA).

PHOSPHATIO DIABETES (see URINE, EXAMINATION OF).

PHOSPHATURIA (see URINE, EXAMINATION OF).

PHOSPHORUS POISONING (see POISONS AND POISONING).

PHTHIRIASIS (see PEDICULOSIS).

PHTHISIS (see PULMONARY TUBERCULOSIS).

PICA (Dirt Eating).—The habit of eating dirt, cinders, sticks, wool off blankets, etc., or of licking paint off toys or whitening off the hearth, is not uncommon in small children of a few months old and upwards. It is normal for a child to put in its mouth all it can lay its hands upon; only when the habit is inveterate can it be regarded as a functional neurosis. Children who are morbid in this respect often show other evidences of a neurotic temperament. As a result of their gustatory proclivities they become pale, thin, and irritable, lose appetite for food, and suffer from intestinal disturbance, the motions being loose and offensive, containing perhaps mucus and blood and the various foreign bodies which have been swallowed. Apart from its occurrence in otherwise normal children, pica is common in imbeciles and idiots, and may be a symptom of mental disease in later life.

Treatment is by persuasion and correction. Small doses of bromide are often beneficial. When intestinal disturbance has been set up, a brisk purge, such as castor oil, is necessary to clear out the source of irritation.

FREDERICK LANGMEAD.

PIGEON BREAST (see CHEST, DEFORMITIES OF).

PIGMENTATION.—Pigmentation of the skin, in white races, occurs under a large number of conditions.

Physiological pigmentation may arise from exposure to wind or sun; it is customary in old age, and is included also among the signs of pregnancy. Apart from the usual areolæ about the nipples and the pigmentation of the

PIGMENTATION

linea alba in pregnancy, the patchy pigmentation of the face ("masque des femmes enceintes," or chloasma uterinum) is well known. A pigmentation of the skin on the fronts of the legs is met with in people who habitually sit in front of the fire.

Pathological pigmentation.—The most important cause is *Addison's disease*. In it the pigmentation is not limited to the skin but affects also the mucous membranes, and may be detected in the mouth and vagina. In the mouth the patches of bronze colour should be looked for on the inner surfaces of the lips, the gums, the palate and the buccal mucosa. In the latter situation the appearance in an advanced case may resemble that normally seen in a spaniel. The patches on the skin are usually of the same dark-brown or bronze colour, and appear first in those places where pigment is normally found, as the face, axillæ and groins, and in the areas subjected to pressure, whether by the collar, braces, belt or other apparel, or by the apposition of the parts. On the trunk and abdomen, especially the lower abdomen, which is frequently thrown into folds, it may occur as irregular transverse bands. Occasionally the pigmentation is almost black, while lighter shades than the customary bronze are occasionally met with. The pigmentation of the mucous membranes is not an absolutely diagnostic sign, for it is sometimes met with in other conditions. A considerable degree of pigmentation is not uncommon in certain abdominal diseases, especially in *tuberculous peritonitis* and *carcinoma*. The explanation is probably the same as that of Addison's disease, and denotes an interference with the chromaffin system. The pigmentation of old-standing cases of *chronic nephritis* may fall into the same group, as may also that of *xerophthalmic goitre*, for the relationship between adrenal secretory disturbance and hyperthyroidism is now known to be intimate. In the subject of *chronic constipation* the face and neck are liable to assume a muddy, unwashed appearance, which may be either general or patchy in distribution.

Pigmentation occurs also in *cirrhosis of the liver*, especially the biliary form described by Hanot, and in that accompanying *hæmochromatosis* (diabète bronzé).

In *ochronosis* the pigmentation is black in colour and affects the cartilages.

A more common variety is that resulting from irritation by pediculi and dirt, seen especially in tramps and known as *vagabond's*

disease. Sun and wind here play their part also.

In *melanotic sarcoma*, in addition to the pigmented growths, a diffuse black discoloration of skin sometimes occurs.

A general bronzing, closely resembling that of Addison's disease, is sometimes met with in *malaria*; several examples were seen in the Salonika army. Among other diseases of tropical and subtropical countries in which pigmentation is found are *leprosy* and *pellagra*.

Two drugs stand out as especially provocative of pigmentation—*arsenic* and *silver*. In the former it is prone to appear on the thorax and spread to the neck and axillæ, but it may resemble the pigmentation of Addison's disease very closely. In other cases it is scarcely distinguishable from ordinary freckles. The associated symptoms of arsenical poisoning should make the diagnosis clear. In silver pigmentation (*argyria*) the colour varies from a light grey to a dark slaty hue; it appears first in the gums, resembling the line of plumbism, and may become general. It is sometimes confined to the area to which the metal has been applied, e.g. the conjunctivæ, or, in the case of silver workers, the hands. Though now comparatively rare, it is occasionally met with, especially among the workers in artificial pearls, who use this metal as a pigment.

Congenital melanoderma is by no means uncommon, but usually takes the form of one or more well-defined discrete patches.

Of many skin diseases pigmentation is an important feature; examples are lentigo, pityriasis versicolor, pigmented moles, urticaria pigmentosa, xeroderma, and xanthoma. The well known "rusty-ham" discoloration peculiar to syphilitic rashes must be included, and also that at the site of hæmorrhages, as after purpura. Many skin lesions, as the rash of varicella and of variola, herpes zoster, and suppurative lesions, if severe or irritated are liable to be supplanted by a patch of pigmented skin when healing has taken place. Similarly, pigmentation may follow lesions artificially produced, as by blisters or mustard plasters.

Rheumatoid arthritis includes among its manifestations pigmentation of the skin. Small spots or smears may be scattered over the body, or there may be larger areas, especially over the face and neck. Its shades are very various, particularly on the face, where the discoloration may be yellowish or dark brown. The forehead often has a burnished appearance in reflected light.

PITUITARY GLAND, AFFECTIONS OF

In *von Recklinghausen's disease* patches of melanoderma are associated with fibromata and plexiform neuromata, or perhaps with tumours whose structure is chiefly fatty.

The greenish discoloration seen in *chloroma* still awaits explanation.

Pigmentation due to the introduction into the skin of *extraneous pigments*, such as occurs in aniline dye workers, in munition workers, and as the result of tattooing, needs only to be mentioned.

That variety of pigmentation known as jaundice is described elsewhere (see JAUNDICE).

FREDERICK LANGMEAD.

PILES (see HÆMORRHOIDS).

PITUITARY GLAND, AFFECTIONS OF.

The pituitary gland, or hypophysis cerebri, is situated at the base of the brain, tucked away in the angle formed by the optic chiasma and the optic tracts, and ensconced in the sella turcica of the sphenoid bone.

From time to time various theories have been held as to its function, and the following important facts are now universally recognized. First, it is essential to life, and removal of the gland, or any cause that determines total cessation of its activities, results in death within a few days. Secondly, it produces internal secretions which are discharged into the blood and give rise to far-reaching effects.

The gland consists of three portions. The anterior and intermediate parts are formed by epithelial cells derived from a prolongation of the buccal mucous membrane, known as Rathke's pouch, and the posterior part is formed by neuroglial cells and fibres that have originated in the brain. The whole gland is suspended from the base of the brain by a stalk or infundibulum, which in man contains no central cavity (Blair Bell).

The functions of the three portions of the gland have been a matter of considerable discussion; but in order to have a sound working knowledge that will explain the various clinical manifestations of pituitary disorders met with, it is best to adopt the theory which seems to be most probable in accordance with our present knowledge. For this we are indebted to those scientists who have, little by little, brought light out of darkness, and while realizing that it is impossible to give due recognition to all in the space available, the names of Oliver, Schäfer, Howell, Swale Vincent, Paulesco, Cushing, and

Blair Bell must be mentioned. The most reasonable view appears to be that put forward by Blair Bell. The granular cells in the anterior and intermediate lobes produce a secretion that is poured into the blood; some of this secretion passes through the posterior lobe before gaining access to the circulation, and is there modified so that it obtains the peculiar physiological properties exhibited by an extract of the posterior lobe. There is no definite evidence in man to prove that the secretion of the pituitary is discharged into the third ventricle of the brain, and so into the cerebro-spinal fluid. The secretion from the anterior lobe is probably concerned with the growth of the body, with deposition of fat, and with sexual activity; that of the intermediate lobe with sugar metabolism; and that of the posterior lobe with the maintenance of blood-pressure and the tone of involuntary muscle throughout the body, and also with the secretion of urine.

The function of the pituitary may be disturbed in two ways: it may produce too little or too much secretion, from any portion. Such disturbances are grouped under the term *dyspituitarism*, the former being known as *hypopituitarism*, and the latter as *hyperpituitarism*. They may be caused indirectly by an alteration in one or other of the ductless glands that produce internal secretions, for the endocrinous glands of the body are all closely related, and disturbance of function in one will cause changes that are probably compensatory in nature in the others. On the other hand, the *fons et origo mali* may be situated actually in the pituitary itself, and take the form of a hyperplasia, or tumour. Again, the pituitary may be compressed by any cause giving rise to an increased intracranial pressure, such as a hydrocephalus.

A. DISORDERS OF THE PITUITARY ASSOCIATED WITH AN INCREASED SECRETION

The effects produced vary with the age of onset.

1. **SEXUAL ABNORMALITIES.**—An excessive secretion (probably from the anterior lobe) leads to a condition of sexual precocity, and a similar condition in girls tends to exaggerate the masculine traits at the expense of those peculiar to their own sex.

2. **GIGANTISM.**—It is believed that over-activity of the pituitary increases the rate at which bone is formed, and if this occurs before the epiphyses have united there is an increase

in the length of the bones of the extremities, and a condition of gigantism results.

3. **ACROMEGALY.**—A similar process is thought to be concerned with this peculiar disease, but the hypersecretion in these cases occurs after epiphyseal union has taken place; often in the final stages the secretion, instead of being in excess, becomes much diminished, and further characteristic changes ensue.

Acromegaly signifies that there is an increase in the size of the extremities, chiefly of the hands. The disease generally occurs in the third or fourth decade, and is more common in females than in males. In the vast majority of cases there is present a pituitary tumour, generally an adenoma or carcinoma, or in some instances a sarcoma.

Symptomatology.—The symptoms of acromegaly are due to the alterations in the secretion, and to direct pressure by the growth. The skeletal changes embrace enlargement of the head, hands, and feet, the most noticeable changes in the skull being increase in size and prominence of the lower jaw and gradual spacing or separation of the teeth. The hands become clumsy and the fingers spatulous. The tongue is larger than normal, and the nose broader and flatter; the skin is rough and thick. There is often an increase in the amount of urine secreted, and in some cases glycosuria. Towards the end of the disease, when the tumour has destroyed to a large extent the pituitary's power of secretion, we find signs of hypopituitarism, the sexual faculties and desires are lost, the glycosuria is replaced by an increased sugar tolerance, and the patient may become extremely obese.

Symptoms due directly to the cerebral tumour are headache, which is often peculiarly severe, and eye signs. Especially frequent is blindness of the nasal half of each retina (bitemporal hemianopia), caused by the tumour either dragging on or compressing those fibres of the optic tract that have decussated in the chiasma. Sometimes the tumour compresses the third nerve on one side, or even the fourth or sixth, giving rise to corresponding oculo-motor paralyses. Towards the end of the disease there is an increased torpor, and finally dementia may ensue, which accounts for the fact that a considerable number of acromegals end their days in an asylum.

The **diagnosis** is made generally with ease, for when once the condition is well established the facies is almost unmistakable. In addition to the signs and symptoms mentioned above,

the X-rays afford valuable confirmatory evidence. In a typical case it is found that the sella turcica, whose normal measurement is 11 to 15 mm. from before backwards and 6 to 9 mm. in depth, is enlarged; further, the clinoid processes, more especially the posterior ones, may be eroded and disappear. The frontal sinuses are often enlarged and the superciliary ridges thickened, in correspondence with the increase in size of the skull. The hands show characteristic changes, there being a peculiar tufting of the bone of the extremities of the terminal phalanges, sometimes with the appearance of small exostoses along the phalanges, which are also widened.

The **prognosis** in this disease is bad. In the majority of cases there is a gradual progression towards the fatal ending, which may be postponed for years; but in some cases with rapidly growing tumours death soon releases the sufferer from his distress.

Treatment.—This embraces both surgical and medical therapy. Surgical aid should be invoked to relieve the symptoms caused by increased pressure in the skull. These may be headache due to stretching of the capsule of dura mater in which the gland is enclosed, or headache and other symptoms of a cerebral tumour caused by increased intracranial pressure, or finally symptoms of an ocular nerve affection due to localized damage by the pituitary growth.

Medical treatment is of value to combat the distant results caused by disturbance of the internal secretion. It is clear that it would be irrational to administer pituitary extract in any form in the early stages of acromegaly, during which there is hyperpituitarism, but in the later stages of the disease, when there is deficient secretion, administration of whole-gland extracts by the mouth in doses varying from 5 to 100 gr., t.d.s., may be useful in relieving the torpor and certain of the sexual and mental derangements.

4. **DIABETES INSIPIDUS.**—A certain number of cases are definitely associated with a pituitary lesion, and by inducing the aid of nervous reflexes all cases can be ascribed directly or indirectly to pituitary disturbance. The following lesions have been demonstrated post mortem, viz. a basal syphilitic meningitis of the interpeduncular space, a basal tuberculous meningitis with a caseous nodule in the infundibulum, a malignant epithelial tumour of the pituitary, a sarcoma, a gumma, and an adenoma. Further, it has been shown that

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irritation of the sympathetic nerves running to the pituitary will cause a polyuria, and that polyuria has followed injury to the base of the skull such as that caused by a bullet wound. The immediate effect of administering extracts of the posterior lobe is to cause a diuresis.

The onset of the disease is most common during early manhood and middle life, and is twice as common in men as it is in women. (See also DIABETES INSIPIDUS.)

stages of acromegaly, or in a basal meningitis or in a tumour-formation. It may also be induced by a reflex stimulation whereby excitatory impulses pass to the gland through the cervical sympathetic system.

The **symptoms** are twofold—first, the presence of a mild glycosuria that readily responds to a diminution of the carbohydrate intake; and secondly, the direct effects of pressure of the pituitary tumour, producing eye signs and very often noise in the head and ears.

Diagnosis.—A patient who complains of failing vision, and is found to have glycosuria, runs a considerable risk of being the subject of an error in diagnosis. The eye signs in pituitary lesions differ from those in diabetes in that ophthalmoplegia is due in the former to pressure effects causing paresis, but in the latter to a toxic neuritis. Retinal changes do not often occur in pituitary lesions, but there is a great liability to hemianopia. In other cases the glycosuria may be found to be associated with the distal changes that we have seen to be characteristic of hyperpituitarism, such as bony enlargements, thick and rough skin, a tendency to masculinity in women, or a raised blood pressure.

The **prognosis** depends entirely upon the nature of the pituitary lesion: the actual glycosuria is of no moment, and is easily controlled by dietetic methods.

The **treatment** consists in limiting the carbohydrate intake, and in treating the cause of the hyperpituitarism in accordance with the lesion, as laid down above.

B. DISEASES ASSOCIATED WITH A DIMINUTION OF THE PITUITARY SECRETIONS

Just as the effects of an increased secretion of the pituitary vary according to the age of onset, and whether or not the epiphyses have united, so in the converse condition the results produced are to a certain extent dependent upon the age of the patient. The most outstanding characteristic of hypopituitarism is sexual inactivity and infantilism, and in the vast majority of cases there is a diminished metabolism with deposition of large quantities of subcutaneous fat.

If the secretion becomes deficient before puberty, a condition of infantilism is established which concerns both the outward and physical characteristics of the body and also the development of the genital glands.

1. In the condition of infantilism known as



Fig. 80.—*Dystrophia adiposo genitalis*: a boy aged 10. (Dr. Langmead's case.)

5. **GLYCOSURIA.**—In certain cases in which on routine examination a patient is found to have sugar in the urine, but true diabetes is absent, there is a lesion in the pituitary body. It is known that excessive secretion from the pituitary lowers the sugar tolerance of the body and leads to glycosuria. The condition is not so severe as true diabetes, for the sugar excreted comes from that ingested and from the glycogen stored in the body. There is not the total derangement of metabolism which occurs in diabetes mellitus, whereby the sugar excreted has its origin as protein and fat. Pituitary glycosuria may therefore be caused by any irritative lesion, such as is found in the early

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the *Lorain type* there is no adiposity ; the child does not grow, so that at first sight an adult has the semblance of a child, but a more careful examination shows that the shape is that of a grown man or woman. The condition and distribution of the hair are those of childhood, the sexual organs are immature, but intelligence is not usually impaired. In a certain number of these cases it has been shown that there is a tumour of the anterior lobe of the pituitary.

The diagnosis that a case of infantilism of this type is due to hypopituitarism is strengthened in two ways : (a) by a radiograph of the skull, showing an enlargement of the bony sella turcica due to a pituitary tumour ; (b) by a hypodermic injection of an extract of the anterior lobe of the pituitary, which causes a rise in temperature of one or more degrees—the so-called “thermic reaction” of Cushing.

2. There is another type of infantilism associated with adiposity and occurring before puberty, known as *Dystrophia Adiposo-genitalis of Frölich*. (Fig. 80.) Its cause is believed to be a lesion of the pituitary, and, though the actual pathology of the disease is obscure, it has been shown experimentally that tying or separating the stalk of the pituitary will produce a similar state in dogs ; the operation interferes with the blood supply of the whole gland, producing an alteration in the cells, chiefly in the anterior lobe, with hypopituitarism. In a certain number of cases the condition has either followed an injury to the base of the skull or has accompanied a tumour in or near the pituitary body.

In a typical case of *dystrophia adiposo-genitalis* occurring before puberty the child is considerably shorter than he should be for his age, is remarkably obese, and is very drowsy and somnolent. Boys are formed on feminine lines, the skin is soft and smooth, the genital organs are in a state of hypoplasia, and the blood-pressure is low. A case presenting the clinical picture here described is sufficiently characteristic for a diagnosis to be made. As confirmatory tests, the carbohydrate tolerance should be determined, when it will be found that there is an increased sugar tolerance ; an X-ray may indicate the presence of some pituitary growth ; and, finally, the Cushing thermic test will be positive.

3. A third variety of hypopituitarism has been described as occurring before puberty, in which the adiposity that we associate with Frölich's type of infantilism is present, but the

skeleton, instead of being under-developed, has suffered the reverse change, and there is some bony overgrowth. The pathology is obscure, but it has been suggested that the secretion of one portion of the pituitary, i.e. the middle and posterior lobes, is deficient, whereas there is some excess of that given directly into the blood from the anterior lobe.

Apart from these disturbances of the pituitary secretion which take place before puberty, a hypopituitarism may begin after adult growth has been attained. There is ~~no change in~~ height, but the configuration of the skeleton tends to assume in the male a ~~feminine type~~. Thus the pelvis becomes broader and there is some tendency to genu valgum. The hands are of an artistic type, with delicate tapering fingers. The skin becomes smooth and soft and often dry, with thinning of the hair especially upon the body, and, in the male, a tendency to the female type of distribution of the pubic hairs. A great deposition of fat occurs, the hips become rounded and assume the full curves of the female, and the breasts may enlarge. The body-temperature becomes subnormal and the blood-pressure is low. There is generally a diminution or total loss of sexual function, and in women amenorrhoea may occur.

The pituitary lesion, if a tumour, may cause pressure upon the optic tracts, or may produce a variety of epilepsy with gustatory or olfactory auras owing to irritation of the neighbouring uncinate gyrus of the brain.

4. There are other states of adiposity which are possibly associated with hypopituitarism ; one to be especially borne in mind is that known as *Adiposis Dolorosa* or *Dercum's Disease*. It is found generally in women. Fatty subcutaneous masses make their appearance and are associated with tenderness, pains in the body, loss of strength, and some degree of mental disturbance. In a certain number of cases there are indications that the pituitary is at fault, and not producing sufficient secretion ; thus the skin is often dry, the temperature subnormal, and the sugar tolerance much raised. Further, there may be localizing signs of a cerebral growth in the neighbourhood of the pituitary. (*Adiposis dolorosa* is described more at length under its own title.)

Treatment of cases exhibiting signs of hypopituitarism.—The obvious treatment is an endeavour to supply to the body the secretions that are deficient, but although in some cases a certain amount of improvement follows

the administration of pituitary extracts, the results have not been so brilliant as those obtained in other deficiency-diseases of the ductless glands, such as myxedema.

No definite active principle has yet been isolated from the pituitary comparable with adrenalin, but there is a substance, hypophysin sulphate, which is said to be a definite chemical compound and which is used in a dilution of 1 in 1,000.

Both dried and liquid extracts are available for therapeutic use. **Dried extracts** are usually prepared from the whole gland and are administered by the mouth. Cushing has devised a method of determining the dose required in cases of hypopituitarism. These, as we have seen, generally show an increased sugar tolerance. An amount of glucose or lactose is given sufficient to produce a temporary appearance of sugar in the urine in a normal individual (about 250 grm. of glucose in a healthy adult), and the dose of pituitary extract is increased until a small quantity of sugar appears in the urine. **Liquid preparations** may be made from the anterior or posterior lobe. *Hypophysin* (Fuhner) is an extract of the posterior lobe, as are also *pituitrin*, *infundibulin*, *infundin*, and *pituglandol*. There is also an *elixir hypophysin* made from the whole gland, of which the dose is 1 to 2 dr. The most marked effects are obtained from the liquid preparations if they are given intramuscularly or intravenously; in subcutaneous injection there is said to be a danger of causing sloughing of the skin from the intense vaso-constriction produced.

The best results are obtained from whole-gland extracts given by the mouth, but large amounts may be required to produce definite changes, as much as 100 gr. three times a day being given in some cases. (For further details, see ORGANO-THERAPY.)

By these means gross torpor and drowsiness may be abolished and sexual function restored, but often the sugar tolerance is only slightly lowered and it is difficult to cause any permanent diminution of weight.

Another method of compensating for a deficient pituitary secretion is by transplantation of the gland; in certain instances the pituitary gland has been transplanted from a recently dead infant into the brain of an adult the subject of hypopituitarism, with beneficial results. Surgical measures may be required in cases of hypopituitarism due to a tumour that is causing local disturbance by pressure.

G. E. BEAUMONT.

PITYRIASIS ROSEA.—The principal feature is an acute papular and circinate eruption with a special distribution and evolution. The etiology and pathology remain obscure.

Symptomatology.—Usually without premonitory symptoms, but occasionally associated with a feeling of general illness, sore throat, or polyglandular enlargement, the eruption makes its appearance as an extensive aggregation of minute pink papules and as circinate patches the periphery of which is slightly raised, the centre occupied by a very thin and partially desiccated pellicle, which can readily be detached; its loose adhesion is evidenced by the wrinkled surface like crinkled tissue-paper, an appearance which is highly characteristic of the disease. The colour of the edge is a vivid pink, which warrants the appellation "rosea"; the centre is much lighter in tint, and is often a pale buff. In about 25 per cent. of instances the eruption begins with a single circinate patch which precedes the general rash by a few days, and is therefore called the "pioneer" or "herald" patch. Sometimes the eruption may resolve itself into a generalized fine desquamation in the areas affected. More commonly the lesions become progressively fainter and more buff in colour, and disappear without leaving any trace. This involution may be quite spontaneous, and is not apparently much hastened by treatment; the average duration of the eruption, six to eight weeks, is remarkably constant, and recurrences are extremely rare.

The *distribution* is so characteristic as to be an important aid in diagnosis. The "vest area" is the general site, with special predilection for the sides of the trunk, and in this position the individual lesions tend to be placed with the long axis of the oblong patch in the oblique line of the ribs. The face and scalp are seldom affected, the hands still less often, the mucous membranes never.

Diagnosis.—Although the disease is not uncommon—occurring in the proportion of about 4 per mille in a general skin clinic—it seems to be frequently mistaken by general practitioners for other diseases, especially ringworm, seborrhoeic eczema, and syphilis. *Ringworm* seldom has so wide a distribution, and fungus can readily be found in the scales, whereas it is entirely absent in pityriasis rosea. The distribution of *seborrhoeic eczema* is different, and there is no spontaneous involution. *Seborrhoeic* scales show abundant presence of poly-

PITYRIASIS RUBRA PILARIS

morphous cocci, bacilli seborrhæa, and bottle bacilli, all absent in pityriasis rosea. From syphilis the clinical differentiation may be difficult, and a Wassermann test may be required to settle it. In syphilis the patient is usually more severely ill, glandular enlargements are more general and pronounced, and the rash is more mixed in type, darker in colour, more scaly, and more deeply situated in the skin.

Prognosis.—The patient may be assured with some confidence that the eruption will persist for a few weeks, disappear without leaving any mark, and never recur.

Treatment.—Since the disease undoubtedly recedes without any treatment, this is of little importance, unless given in mitigation of symptoms such as itching, which in a few cases is troublesome, and should be dealt with by avoidance of exciting diet and stimulants, by local antipruritic lotions, and by moderate purgation. I do not believe, personally, that any treatment cuts short the duration of the disease, but there is good authority for recommending salicin in 15-gr. doses three times per diem; daily alkaline baths; or mild antiseptic treatment such as is used by Jamieson, who adds a few teaspoonfuls of Condy's fluid to the bath and then anoints the body with a 5-per-cent. sulphur ointment in vaselin.

E. GRAHAM LITTLE.

PITYRIASIS RUBRA (see DERMATITIS EXFOLIATIVA).

PITYRIASIS RUBRA PILARIS (Devergie) (*syn.* Devergie's Disease; Lichen Ruber Acuminatus (Hebra-Kaposi).—A rare disease of the skin, presenting clinical resemblance to psoriasis on the one hand, and to lichen planus on the other.

Etiology.—The causation of the disease is at present unknown. Suggestions have been made emphasizing its connexion with psoriasis and lichen planus on the one hand, and with certain chronic infections, such as tuberculosis, on the other hand. Reaction to tuberculin of some patients and the superficial resemblance of the disease to such a state as lichen scrofulosorum have given a certain interest to the latter hypothesis. No definite knowledge as to causation is, however, at our disposal. The disease occurs in both sexes, and commences not infrequently in young adults, or even in children.

Pathology.—The microscopic anatomy of

the lesions has been carefully and frequently studied. The main feature of this skin affection is great and almost universal hyperkeratosis. The true horny epithelium is increased noticeably over the areas where the scaly part of the eruption is seen, and still more so in the pilosebaceous follicles of the skin. The horny epithelium of the pilo-sebaceous follicle is increased to such a degree that the infundibulum of the follicle is dilated and filled with horny cells entangling the fine hairs of the skin, causing the papular projection on the surface so characteristic of the disease. The amount of hyperemia varies, but is often considerable; dilatation of the cutaneous capillaries is seen on microscopical examination and is associated with slight cell infiltration in the corium, especially in the papillæ, which may be swollen and flattened in the neighbourhood of the hair-follicles.

Symptomatology.—The disease usually affects the whole surface, but in varying degrees. Some parts, as the face, may show only a certain amount of erythema of the slightly thickened skin, with a tendency to scalliness. Other parts, such as the scalp, may show similar conditions with scalliness more pronounced. In still other areas the slightly cedematous and thickened skin is covered with a finely adherent white scale, sometimes described as being "plaster-like"; these areas are noticeably the knees and elbows, and certain other parts affected by psoriasis. It is for this reason that the disease has been mistaken for psoriasis; the scale, however, is much finer, usually more closely adherent, and though it falls off usually as a fine powder it is not detached in the form of the large flakes so characteristic of the desquamation of psoriasis. It will be noted that the feature of hyperkeratosis is present in all these lesions. This symptom is, however, most pronounced in the areas of the skin where the characteristic follicular lesions of the disease occur. Follicular hyperkeratosis raises the mouth of the follicle as a papule, sometimes definitely acuminate, but usually flattened or rounded as a result of friction. The mouth of the follicle can generally be seen in the centre of the papule. These lesions may involve the pilo-sebaceous follicles over extensive areas, such as the backs of the hands and forearms, the outer surface of the buttocks and thighs, the knees, and elsewhere. A peculiar aspect of the skin is thus produced, which has caused it to be likened to shagreen; it gives the feeling and something of

the appearance of a nutmeg-grater. A general erythema is noticeable in these shagreen-like areas, but usually no definite special inflammatory margin round the horny papules is perceptible to the naked eye. The natural folds of the skin on the flexures and in other situations are much emphasized as the result of slight inflammatory oedema and increase of the horny layer of the epithelium. Attention is often drawn to the appearance of the follicular lesions on the dorsal aspect of the hands and fingers. The individual follicle may be raised, and its little horny plug frequently has a black point in the centre, marking the orifice of the duct and the retention of foreign material. There seems to be little disturbance of health during the evolution of an attack, though febrile symptoms have been observed. The disease persists for many years, sometimes with accessions of severity. There may be considerable periods of improvement, approaching even to cure. As the cases of this disease are so rare and so long in duration, it is not easy to follow them throughout their whole course; apparently some of them improve to such an extent as to become practically well. On the other hand, there appears to be a definite risk that this condition may pass into a state of general exfoliative dermatitis, when the results to the patient are naturally very much more hazardous. Severe forms of exfoliative dermatitis have supervened on pityriasis rubra pilaris.

Treatment.—The treatment most often adopted for this disease proceeds on the same principles as that for psoriasis—soap baths and frictions, followed by the application or inunction of ointments containing exfoliating drugs, such as salicylic acid or resorcin, or drugs giving such special reactions as the varieties of tar, oil of cade, or chrysarobin. But even well-considered efforts on these lines have not given satisfactory results. Probably the most satisfactory local means of treatment are those of an emollient or soothing nature only, such as warm baths, followed by the use of a cream containing small amounts of salicylic acid sufficient to produce slight desquamation, and corresponding usually to the strength of the ointment of the British Pharmacopœia. Simple emollient preparations such as cold cream, and especially glycerinated applications in the form of pastes or salves, are often most beneficial. It should be recollected that patients suffering from this disease are apt to deteriorate in health, so that attacks of tuber-

culosis may supervene, and, further, that the more strenuous forms of treatment with drugs, such as chrysarobin, tar, or the phenyl derivatives, may induce exfoliative dermatitis, with serious consequences.

JAMES GALLOWAY.

PITYRIASIS VERSICOLOR (see *TINEA* (PITYRIASIS) *VERSICOLOR*).

PLACENTA PRÆVIA (see *HÆMORRHAGE*, *ANTE-PARTUM*).

PLACENTAL POLYPI (see *UTERUS*, *POLYPI* *OF*).

PLAGUE.—An acute febrile disease, caused by the *Bacillus pestis*, usually accompanied by a bubo, and frequently terminating fatally amongst Orientals. It is a disease of very great antiquity. It was certainly known to the ancient Egyptians. A virulent epidemic known as the Black Death swept over Europe in the fourteenth century of the Christian era. The great Plague of London in 1665 was the final outburst of an epidemic that began seventy years before. The disease was imported into Goa, in India, in the seventeenth century, and practically ruined that city. At the end of the nineteenth century a recrudescence occurred, the original focus of infection being Southern China and Hong Kong. It was carried by ships to India, and thence to Australia and South Africa, and many other parts of the world, including Portugal and the British Isles.

Etiology.—The *Bacillus pestis* (Fig. 81), discovered by Yersin, is a small oval bacterium which shows distinct bipolar staining and, when cultivated in broth, forms what are known as “stalactite” cultures. It is very fatal to guinea-pigs.

It cannot be too strongly insisted upon that plague, as we know it, is a disease of rats. But the conditions necessary for the communication of the disease to man are almost always present in many portions of the globe, so that in these countries plague has come to be looked upon as a common human malady. This is the key to the understanding of the method of spread of the disease. The fundamental factor in epidemics of plague among human beings is the symbiosis of men and rats. Without this there would be no human plague, and in countries where rats and human beings do not occupy the same house, or rarely do so, the disease will never make any serious headway. We shall see later that this statement

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requires some slight modification, because there are two kinds of plague, each with its own method of spread, but the above is certainly true of bubonic plague.

Plague is an extremely fatal disease among rats and kills them off in thousands. When an infected rat is about to die, plague bacilli are present in the peripheral circulation. Rats always harbour a certain number of fleas in their fur. These insects bite the sick rat and suck into their stomachs the blood and with it the plague virus. When the rat dies of the disease all the fleas leave the body as it turns cold, and seek another rat, and in this way the malady is spread from rat to rat. Should no other rat be available, which is likely to be the

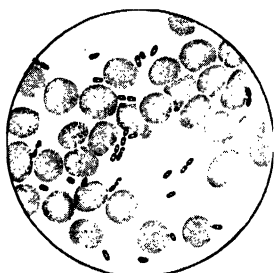


Fig. 81.- *Y. pestis* in peripheral blood in septicæmic plague. $\times 500$. (Microphoto: Dr. J. Bell.)

case when an epizootic is at its height, the fleas are driven by hunger to bite human beings, and the person bitten develops the disease in a few days. Fleas remain infected and capable of disseminating the disease for about a week after infection, but the length of time and the power of passing on the infection depend on the temperature of the atmosphere. Thus, if infected fleas are put into a cold chamber and the temperature is reduced below 50°F ., they very rapidly lose the power of passing on the disease.

The foregoing is a correct picture of the method of spread of ordinary bubonic plague. A few further points which are of great interest and importance must be very briefly stated. These are: (1) The greater the number of rats present per unit of the human population, the greater is the likelihood of plague amongst men. (2) The greater the number of fleas per rat, the greater the rapidity with which the disease will spread from rat to rat, and the greater the

likelihood of men being infected. (3) Any climatic condition which (a) favours the multiplication of fleas, and (b) is near the optimum temperature for rendering fleas infective, greatly increases the possibilities of an epidemic, when once the disease has been introduced. This point has been carefully investigated by the Plague Commission, and it is found that a damp climate of equable temperature of about 70° to 80°F . is most favourable to flea multiplication, and that fleas remain infective longest at a temperature of about 70°F . (4) The factor which tends to increase the number of rats in any particular place is the quantity of food available. Thus, towns with bad surveyancy, with a great deal of rubbish, food refuse, etc., lying about, have always a large rat population. (5) The custom, so common in many countries, of storing grain and other foods in bulk in the dwellings of the people, is a powerful factor in aiding the spread of the disease among men. Not only does it provide an ample food supply for rats and thereby increase their number, but it attracts some varieties into the houses that might remain outside in the fields, and results in rodents living in very close contact with human beings. (6) The kind of rat which inhabits a town is important. There are three chief varieties found in houses (besides the field rats, which only very rarely stray into buildings); these, on account of their natural habits, are not equally liable to spread plague. The three species are the black rat (*Mus rattus*), the brown or Norwegian rat (*Mus decumanus*), and the bandicoot (*Nesokia bengalensis*).

The first of these—the black rat—is the true house-dweller; it very seldom lives in the fields, and never goes far from human habitations. Formerly it was very common in all countries, but the Norwegian rat has driven it out and greatly reduced its numbers in many parts of the globe. The brown rat is a stronger, fiercer animal, and more enterprising and aggressive; it will live wherever food is plentiful, and frequents houses in India and other countries, but does not live in them exclusively; it will live in the fields equally well at certain times of the year, and in England lives almost entirely in sewers. The bandicoot is a large solitary rat that drives away both other varieties wherever it is numerous. It is a house-dweller, but there is never a swarm of bandicoots in one house. They are very susceptible to plague. If plague be introduced into a town inhabited by any variety of rats, a certain number of human cases

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will result, the more violent epidemics being usually associated with the black and the brown rat. In all countries where the conditions enumerated above obtain, and where for two or three months of the year the climatic conditions are suitable, epidemics will always occur if the disease is imported, until the rat population is greatly reduced in number, or all susceptible rats have been killed off, or until the people themselves keep rats out of their houses.

Varieties of plague.—There are two main varieties of plague, (a) bubonic, (b) pneumonic. Some authorities give a much more elaborate classification, and include such varieties as septicæmic, algid, fulminating, ambulatory, etc., but such a classification merely records a prominent symptom or feature met with. In early epidemics both the main varieties usually existed side by side, the bubonic outnumbering the pneumonic, however, by 20–25 to one. Occasionally both small and large outbreaks of pneumonic plague alone have been recorded.

Ordinary *bubonic* plague is contracted by the bites of infected rat-fleas. In 95 per cent. of the cases a bubo or an inflamed lymphatic gland can be found by careful examination. The infection is introduced under the skin by the flea, and the nearest lymphatic gland attempts to resist the invasion; if it is unsuccessful a general septicæmia results and the patient dies; if, on the other hand, the resistance is sufficiently stout, the gland usually breaks down, forming an abscess, and the patient recovers.

Pneumonic plague, on the other hand, is spread directly from a patient to contacts, and is one of the most infectious diseases known. I have seen instances in which every person who came near a patient suffering from pneumonic plague has contracted the disease and died; this was in the late 'nineties, but the malady does not appear to be so deadly now. Once established, pneumonic plague is not dependent either on rats or on rat-fleas for its spread, but is passed from patient to patient in the same way as diphtheria. As its name suggests, the disease is a plague pneumonia; the patient blows millions of virulent bacilli into the atmosphere with each fit of coughing, and those near him, by inspiring the virus, usually contract the disease.

It may be asked, How does the first case of pneumonic plague occur, seeing that flea-bite infection produces only the bubonic type? The answer is not by any means easy, but there is little doubt that in virulent epidemics, in a

very susceptible community, a certain small number of flea-infected cases develop the pneumonic type, and when once these are established, spread of the pneumonic variety is easily accounted for. In these pneumonic cases, probably—the suggestion is made with great reserve—the infection represents a dose of very virulent organisms, the poison rapidly invading the capillaries and the blood-stream generally, instead of being confined to the lymphatics for some time. It is obvious that, in the ordinary way, a virus introduced just under the skin would be carried by the lymphatics to the nearest gland; on the other hand, should it pass directly into capillaries or veins the most natural result would be a primary pneumonia.

Incubation period.—The incubation period may be as short as two days; three to four days is probably about the normal period; it may be as long as seven days.

Symptoms. Bubonic plague.—The onset is usually sudden. The patient shivers, his temperature rises to 102° or 103° F., and he complains of a painful lump or bubo in the groin, armpit, or neck. On examination it is found that his eyes are injected and suffused, and he rather resembles in appearance a case of acute influenza. He is obviously seriously ill, and is often sleepy and difficult to rouse. The pulse is very rapid (120 or more), thin, thready and soft, the heart-muscle being clearly much affected by the toxæmia. The patient complains of great pain in the bubo and will not allow it to be touched. The bubo is a hard, brawny mass; only in very mild cases can the gland itself be felt. It is unlike the gonorrhœal or syphilitic bubo because it is much bigger, and moreover is usually situated in Scarpa's triangle (when the femoral glands are involved) and not in the inguinal region. Although it is hard and brawny to the touch, there is no sign of suppuration. In bare-legged and bare-footed races the groin is the usual site, the armpit next, while the neck is comparatively rarely affected. The other organs—liver, spleen, and intestinal tract—appear to be normal. A little albumin in the urine is not unusual. The patient's condition then gets progressively worse: he becomes dull, delirious, and comatose, the heart-muscle gives out, and death follows on about the third or fourth day. This description applies chiefly to Orientals. In the European the temperature remains high for about four to six days and then gradually comes down, the bubo either subsiding or suppurating

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and the patient frequently recovering. Should the disease not take a favourable turn he slowly sinks and dies of heart failure.

Occasionally a bubonic case develops a secondary pneumonia. It is usually a bad sign, and the cases often end fatally, but it is not of very common occurrence. The lung trouble generally begins from the fourth to the sixth day of the disease; just when one expects the patient to show considerable improvement in general condition he starts coughing, bringing up a very red sputum; the temperature, which was showing signs of falling, now mounts up again, and the patient usually succumbs after a couple of days.

Pneumonic plague.—The first thing that one notices about a patient suffering from pneumonic plague is that he is profoundly ill and has all the appearances of one who may die in the course of a few hours. His pulse is extremely feeble and very fast. The fever is high—103° F. or more. He coughs weakly, bringing up a sputum which is bright red in colour. Some have described this as like prune juice, but in many cases it is practically blood-red. Examination of the chest reveals very few unusual physical signs. Occasionally quite small patches of dullness can be found, but this is unusual; moist râles are heard at the bases and over the chest generally, but the striking feature is the scantiness of gross lesions in the chest, considering the patient's desperate condition. It is very rare for large patches of consolidation with its accompanying physical signs, as in croupous pneumonia, to be observed, possibly because the patient dies before they can form. Plague pneumonia is a capillary pneumonia. Practically all cases of primary pneumonic plague die; some last as long as three days, but the majority die within forty-eight hours from heart failure.

Atypical forms.—There are atypical cases that are less easy to diagnose. The commonest of these is what—for want of a better name—is called *septicæmic* plague. The name is obviously unsatisfactory, because all fatal cases are septicæmic. In this variety there is no bubo and no pneumonia. There is nothing whatever to assist the diagnosis; the fever, the pulse, and the appearance of the patient are as already described. In the early days of the present epidemic these cases were fairly common; they all ended fatally in a few hours.

Algid plague is exactly like cholera in its symptoms and physical signs. It is quite

impossible to diagnose the disease without laboratory assistance, but fortunately it is very rare.

Ambulatory plague is the name given to a very mild form of the bubonic variety in which the patient has a bubo and a little fever, and walks about as usual.

Fulminating plague is simply a more than usually virulent variety of septicæmic plague. I have seen children playing about perfectly well at 7 a.m. and at 11 o'clock they were dead.

Prognosis.—In early epidemics with a very susceptible population 85 to 90 per cent. of the bubonic and all the pneumonic cases died. Towards the end of the epidemic the proportion of recoveries may rise to 50 per cent., and the average case-mortality for the whole epidemic may be as low as 60–70 per cent. Often the first favourable symptom in any case is evidence of suppuration in the bubo. Amongst Europeans the death-rate is much lower; in an Australian outbreak with a largely European-derived population it was not higher than 28 per cent.

Treatment.—There is practically no specific remedy. Many drugs have been tried, such as creosote, carbolic acid, and perchloride of mercury, but none has had the slightest effect within my experience. The only thing to be done is to feed the patient with small quantities of easily digestible food and give stimulants. I have used a mixture of milk and brandy or milk and rum with good results in large epidemics when short-handed; the mixture is palatable, cheap, can be given by the friends of the patient, and is suited to the Oriental. Some authorities consider that *strophanthus* and *digitalis* can also be given with advantage to help the heart, but they do little good in the case of Orientals, and Europeans do not often require them.

Prophylaxis.—While none of the curative serums, such as the Yersin-Roux serum, has given favourable results, Haffkine's prophylactic vaccine is a thoroughly established prophylactic agent and has been used on a very large scale. The whole population should be inoculated with it when an epidemic threatens (that is, when dead rats are found in any numbers) or is in progress. Inoculation greatly reduces not only the danger of contracting the plague, but also the chances of a fatal result should the disease be contracted.

Outbreaks of plague in England are only likely to occur in seaport towns, since infection

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must come from outside. A ship may bring infected rats, which may escape ashore and spread an epizootic among the shore rats. If this occurs it does not by any means follow that an epidemic among human beings will ensue, because the rats in this country mostly live in the sewers and not in the house, but isolated cases in man are likely in cities. If human plague does supervene, a rat campaign should at once be started; the patient's clothes and bedding should be disinfected with steam in order to kill any rat-fleas, and the premises whence the patient came may be disinfected, but too much reliance should not be placed on these measures, because the probabilities are that the fleas are all dead or cease to be infective by the time the case is reported. Should a medical man come across a doubtful case of plague, he should at once communicate with the Health Authorities, and special bacteriological tests should be applied to verify the nature of the disease. The patient is almost certain to be a sailor or docker or in some way connected with shipping.

As true bubonic plague is never spread from man to man, no particular precautions need be observed by nursing staffs in these cases. Pneumonic plague, however, is quite the most dangerous of all diseases that medical men and nurses are called upon to face, and the utmost care must be observed in all such cases. The patient should be treated in the open air. In India I was able to arrange for all my cases to be treated under trees or in tents. In England the small open huts used by tuberculous patients should be utilized. The nurse and medical man must wear masks of lint and wool over the nose and mouth; in the Manchurian epidemic it was clearly proved that this greatly reduced the chances of infection. A spray may be kept constantly going, but open-air treatment is safest, simplest, and best.

The nurse should be instructed to stand aside or behind the patient during a fit of coughing. All who came into close contact with the patient before the nature of the disease was suspected should be removed to hospital and segregated, and all arrangements made at once for treating them, for, if the case is one of pneumonic plague, the probabilities are that they will sicken with the disease in a few hours' time.

W. W. CLEMESHA.

PLEURA, NEW GROWTHS OF (see
LUNG, MALIGNANT NEW GROWTHS OF).

PLEURISY

PLEURISY. Etiology.—Inflammation of the pleural membrane is probably in all cases due to bacterial infection, and few of the ordinary pathogenic microbes are incapable of giving rise to it. Many of them usually set up a purulent inflammation (see EMPYEMA), but this article deals only with so-called simple or non-purulent pleurisy. By far the commonest cause of this variety is the tubercle bacillus. It is true that the great majority of sero-fibrinous pleural effusions are sterile, but if large quantities of the effusion or of the centrifugalized sediment are injected into the peritoneal cavity of guinea-pigs, tuberculosis follows in a considerable proportion. Although tubercles are very difficult to find on the surface of the inflamed pleura, as they are masked by the inflammatory exudation, they may be detected by careful search, especially between the lobes of the lung. Moreover, a large proportion of patients who have had simple pleurisy subsequently develop phthisis.

Pneumococci and streptococci, which as a rule cause empyema, occasionally give rise to a simple pleurisy, especially perhaps in cases of Bright's disease. The pleural effusion which often complicates, or constitutes a sequel of, acute pneumonia is usually purulent or soon becomes so; but in adults it sometimes remains clear, though very rarely in children. Acute rheumatism is probably a cause of a certain number of cases of simple pleurisy in children, especially in association with rheumatic pericarditis, but the question cannot be regarded as finally settled; certainly the cases are not numerous and the pleuritic effusion is rarely large. Pleurisy is not infrequently met with in connexion with a pulmonary infarct involving the surface of the lung, but the bacteriology of these cases is uncertain. It may be noted that a unilateral pleural effusion in a patient with advanced heart disease is more likely to be due to an infarct than to be dropsical in nature. Occasionally a simple pleurisy is set up by extension of inflammation from the abdomen, but such cases are usually purulent. It may also be due to trauma, as when the pleura is damaged by a fractured rib. A purely secondary pleurisy, often not leading to any recognizable effusion, is common in association with various diseases of the lung, particularly with acute pneumonia and in the course of practically every case of chronic phthisis. Intrathoracic growths frequently give rise to pleural effusion.

The all-important practical point, however,

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to remember about the etiology of simple pleurisy is that any attack, either with or without effusion, for which no other adequate and obvious cause can be found, is in all probability tuberculous in nature, and should be treated as such. Sometimes it seems as though the disease were distinctly due to a chill, or perhaps to an attack of influenza; nevertheless, these alone would almost certainly not have given rise to it unless the tubercle bacillus had been present also. Pleurisy, in fact, bears much the same relation to chill as does tuberculous disease of a joint or of the spine to the slight trauma which so often appears to cause it. Tuberculous pleurisy may occur at any age, but is most common between 20 and 40.

Morbid anatomy.—The disease is usually unilateral. The first change is an increased vascularity of the surface of the pleura, which is soon followed by the appearance of lymph. The process may stop here (dry pleurisy), any fluid effusion being absorbed almost as soon as it is poured out, and more or less extensive adhesions forming quickly between the inflamed pleural surfaces. In cases of active phthisis these adhesions are of the utmost importance in preventing the occurrence of pneumothorax, which would otherwise be almost inevitable.

In severer cases the effusion is poured out more quickly than it can be absorbed, and there collects the familiar sero-fibrinous effusion which is often erroneously spoken of as "serous." This is a yellowish, alkaline, highly albuminous fluid, with a specific gravity of 1015-1025. It is fairly clear, but usually contains floating flakes of fibrin. Like other inflammatory, as contrasted with dropsical effusions, it usually coagulates spontaneously after withdrawal from the chest, but may not do so if the inflammation has been of a mild type. In the latter case the specific gravity of the fluid is lower, it contains much less albumin, and approximates in its characters to those of the fluid in a hydrothorax. An inflammatory effusion contains a variable number of leucocytes, and in tuberculous cases lymphocytes greatly predominate—an important diagnostic point. Occasionally the effusion is hemorrhagic; this is sometimes regarded as suggestive of malignant disease, but is not very uncommon in cases of ordinary tuberculous pleurisy. Much rarer is a chylous effusion, which usually indicates some obstruction of the thoracic duct.

As the fluid accumulates, a corresponding

area of lung becomes collapsed. The alveoli are not uniformly diminished in size throughout the lung, but there is complete collapse of a certain number only, the rest probably remaining unaffected. Gradually the lung is rendered completely airless. If the effusion still continue to increase, it now exerts a well-marked positive pressure and expels blood as well as air from the lung, which becomes dry and entirely collapsed round its root, close to the spine, and of a slate-grey tint (carnified). In such cases the liver or spleen, according to the side affected, is pushed down, owing to downward displacement of the diaphragm. The heart is displaced to the unaffected side by the unantagonized elastic traction of the healthy lung, and this displacement is much more considerable if the effusion be sufficiently large to lead to complete collapse of the lung and to positive pressure upon the contents of the mediastinum. It should be noted that whereas the heart is usually displaced even by an effusion of merely moderate amount, the liver or spleen is pushed down only when the pleural cavity is full of fluid.

Symptomatology.—In many cases the disease begins acutely with shivering and a sharp rise of temperature, but it is more important to note that the onset may be most insidious, with merely indefinite pains, referred to the side of the chest or to the abdomen, and slight general malaise. In some of the latter cases, though certainly not in all, there is no recognizable effusion, but most extensive adhesions and thickening of the pleura may result. Except in the mildest and most insidious variety, there is some fever, intermittent or remittent in character, and rarely rising above 102° or 103° F. Its duration varies from a few days to several weeks, and its subsidence is gradual. It is important to remember that pleurisy is one of the causes of so-called "continued fever." The pyrexia is, in fact, that of a tuberculous disease. Symptoms of general malaise, such as quickened pulse, loss of appetite, and furred tongue, proportional to the height of the temperature, are present but call for no special description. Of the local symptoms, by far the most important is pain with inspiration and coughing, often associated with tenderness. It is usually felt below the nipple or in the lower axilla, but may be referred to the abdomen even as low as the iliac fossa, and hence, if on the right side, it sometimes leads to a diagnosis of appendicitis, sufficiently definite to result in opera-

tive interference. It generally subsides on the third or fourth day, as adhesions begin to form or the inflamed surfaces become separated by effusion. The cough is either dry or associated with a little expectoration. Any *dyspnœa* present is due, at first, to the shallow breathing enforced by the pain on inspiration; later on, its severity is determined by the size of the effusion and still more by the rapidity with which it is formed. As the effusion increases, the patient tends to lie on the affected side so as to give the sound lung the freest possible play. When pleurisy occurs in old people, or is secondary to Bright's disease, the symptoms may be almost latent, even though a large effusion be present. Conversely, the symptoms are often unusually severe in previously robust subjects. During recovery from an attack and while adhesions are forming, some discomfort or even dragging pain may be complained of on the affected side.

Physical signs.—In the early stage, when pain is severe, there may be deficient movement and consequently weak breath-sounds on the affected side, but the only distinctive sign is pleural friction, which is heard best in the axilla or below the angle of the scapula. As it is usually audible only at the end of inspiration, it is often very difficult to hear in the most acute cases, for the pain to which the rubbing together of the acutely inflamed pleural surfaces gives rise is so severe that the patient either cannot or will not take a sufficiently deep breath. In less acute cases it is heard much more easily, perhaps both with inspiration and with expiration, is of a coarser character, and may often be felt (friction fremitus).

When effusion develops, the signs become more pronounced. The affected side is more or less immobile and somewhat larger than usual; by means of a cyrtometer it may be shown to be more rounded than the other side. It must be remembered that normally, in right-handed people, the semi-circumference of the right half of the chest is somewhat greater than that of the left. Bulging of the intercostal spaces occurs only in the rare instances in which the effusion is sufficiently large to cause complete collapse of the lung with positive intrapleural tension. Vocal fremitus is diminished or lost, and the heart's apex beat displaced more or less towards the unaffected side, especially when the left pleura is affected. On percussion, there is an absolutely dull note over the effusion, with greatly

increased resistance, due partly to the fluid and partly to the collapsed lung; the displacement of the heart can also be further demonstrated. The dullness begins at the posterior base and gradually extends upwards and forwards, often rising highest in the axilla, when the pleural cavity is partly full. When the dullness extends to about the fourth rib in front, a more or less tympanitic note (skodaic resonance) can often be elicited below the clavicle; this persists longest in a triangular area below the inner end of that bone, and finally disappears as the effusion rises to the top of the pleural cavity. The dullness varies very little with the position of the patient, although this in some measure determines its situation whilst the effusion is being poured out, for if the patient be then lying on his back the dullness will be more extensive posteriorly than if he had been sitting up or getting about. Very different is the dullness of an effusion associated with a pneumothorax, for this shifts as freely with the position of the patient as does that of an ascitic accumulation in the abdomen. A triangular area of relative dullness (Grocco's triangle) can often be detected beside the spine on the healthy side; it disappears if the patient lies on the affected side. The apex of the triangle is directed upwards and reaches to about the upper level of the effusion; its base extends directly outwards from the spine, along the normal lower limit of lung resonance, for 1-2½ in. The breath-sounds over this dull area are generally weak.

The auscultatory signs of a pleural effusion are less reliable than those afforded by the other methods of examination. The usual condition is diminution, culminating in complete loss, of the breath-sounds and of vocal resonance over the fluid, but there are many exceptions, especially in children. It is not at all uncommon to hear bronchial breathing and bronchophony, sometimes distant and sometimes loud, especially in cases in which a large effusion compresses the lung and so renders it a good conductor of vibrations between the still patent air tubes and the fluid. The voice sound has often a peculiar bleating character (ægophony) which points strongly to the presence of fluid, especially perhaps of a comparatively thin layer, but it is so uncertain that no importance must be attached to its absence. At the apex of the lung the auscultatory sounds may be distinctly cavernous in character—blowing or

amphoric breathing, bronchophony, and cavernous crepitations; and, in view of the fact that the pleurisy is probably rightly regarded as tuberculous, the unwary observer may be led to diagnose with much confidence the presence also of advanced phthisis. Disillusionment comes when, a few weeks later, the effusion having subsided, the signs at the apex are found to have become perfectly normal. Over the unaffected side the breath-sounds are usually extremely harsh or compensatory. The point of maximum intensity of the heart-sounds may help in determining the position of that organ, particularly in those cases in which the apex beat is situated behind the sternum. A systolic murmur is occasionally heard over the base of a much displaced heart. As an effusion is absorbed and the pleural surfaces once again come into contact, friction often returns. This so-called "redux" friction has usually a coarse, leathery, or creaking character; it is heard both with inspiration and expiration, and is not associated with pain.

Diagnosis.—Since many cases of tuberculous pleurisy come on most insidiously, with but few and indefinite localizing symptoms, it is always one of the conditions to be especially looked for in all cases of obscure febrile illness, particularly in patients who are supposed to have had an attack of influenza and in whom the temperature has not fallen to normal after a few days. In post-mortem examinations it is common to meet with extensive pleural adhesions, although no history suggestive of any attack of pleurisy had ever been obtained during life.

In the early stage of acute pleurisy it may be difficult to distinguish it from *intercostal neuralgia* or from so-called *rheumatism of the intercostal muscles* (pleurodynia). The discovery of friction settles the diagnosis, but, as already pointed out, it is often very difficult to elicit friction when associated with severe pain. If there is any rise of temperature it is wise always to regard the condition, provisionally at least, as pleuritic, but a normal temperature does not exclude a mild pleurisy.

In the diagnosis of pleural effusion many difficulties may arise. In physical examination special importance should be attached to displacement of the heart. Radiography will often give important information, but in many instances only an exploratory puncture will finally decide whether an effusion is present or not. Even then there is always the possibility that the needle of the syringe may get blocked

by a flake of lymph or, very occasionally, that the fluid may be inaccessible owing to its being encysted between the lobes of the lung.

It is often difficult to distinguish between *acute pneumonia* and *acute pleurisy*, the more so as pneumonia is nearly always associated with some pleurisy. We are prone, in physical examination of the chest, to attach undue importance to auscultation and not sufficiently to note and weigh the indications afforded by other methods of investigation; in the diagnosis of fluid, at any rate, the results may be disastrous. Bronchial breathing and bronchophony may be heard, as already mentioned, over an effusion, and suggest the presence of consolidation rather than of fluid, but with the latter the heart is probably displaced. Moreover, the symptoms are quite different from those of pneumonia, unless, indeed, the pleurisy be due to a pneumococcal infection, which, in some rare cases, would seem to occur apart from any disease of the lung. On the other hand, a small pleural effusion may mask completely the characteristic physical signs of a pneumonic consolidation, and the diagnosis must then turn on the presence of the special symptoms of pneumonia, such as sudden onset, high fever, rapid breathing, rusty sputum, and herpes on the lips.

After an attack of pleurisy there frequently remains a good deal of *unexpanded lung and thickened pleura*, the physical signs of which may be very similar to those of a small effusion—diminution or absence of vocal fremitus, dullness and increased resistance, weak breath-sounds, and diminished vocal resonance. In the case of an effusion, however, the heart is displaced towards the sound side, while the measurement of the affected side is at least as great as, and probably greater than, that of the healthy one. In a patient with thickened pleura these conditions are reversed; the diseased side is contracted, and the heart is drawn over towards it. There are, however, doubtful cases in which it may be necessary to resort to puncture before a correct decision can be reached.

A *new growth* in the lung or mediastinum may give rise to signs and symptoms almost indistinguishable, for a time, from those of simple pleurisy, particularly when, as not infrequently happens, it is actually attended by a pleural effusion. When the fluid is withdrawn, suspicion may be aroused by its being very much bloodstained (though this is by no means necessarily indicative of malignant

disease), and occasionally cancer-cells may be found in it. Often, however, the diagnosis first becomes evident when less fluid than was expected is drawn off, the physical signs remaining almost unaltered, although further attempts to find more fluid prove futile. Sometimes the pleural cavity refills with great rapidity, possibly in a few hours. Extension of dullness across the middle line always favours the diagnosis of a growth (see MEDIASTINUM, AFFECTIONS OF). A *hydatid cyst* in the lung may give rise to signs so closely simulating those of a pleural effusion that the diagnosis can only be made by the withdrawal and examination of some of the fluid. Unfortunately, the puncture of the cyst may lead to its rupture into the lung and so to sudden death of the patient by "drowning."

Morbid conditions below the diaphragm may push up that muscle, lead to collapse of the base of the lung, and thus closely simulate a pleural effusion. Such conditions are abscess or hydatid in the upper part of the liver and subphrenic abscess. Radiography may be most helpful in such cases. Otherwise, the true situation of the lesion may be indicated by the fact that the lung resonance near the spine extends down almost to the normal level, whereas the dullness rises high in the axilla. It must not be forgotten that disease below the diaphragm may give rise to effusion above it.

A *pericardial effusion* may lead to extensive collapse at the base of the left lung, which may give rise to physical signs simulating those either of pulmonary consolidation or of pleural effusion. An exploring needle may easily be passed through a collapsed lung into a pericardial effusion, and the fluid withdrawn regarded as pleural in origin. It is not uncommon for pleural and pericardial effusions to occur together, in which case the latter is very likely to be overlooked.

The question as to the *probable size of a pleural effusion* is answered chiefly by the extent of the physical signs, but these may be very misleading; sometimes the quantity of fluid is less than the signs would lead one to expect, and sometimes the reverse is the case. It happens occasionally that even a fairly large effusion (perhaps two pints) does not give rise to any recognizable displacement of the heart.

Finally, the *nature of the effusion* has to be determined—is it dropsical, inflammatory, or purulent? A dropsical effusion (hydrothorax)

occurs as part of a general dropsy, is bilateral, and therefore cannot attain a large size. It is true that sometimes a unilateral effusion occurs together with a cardiac dropsy, but such a condition should always arouse a strong suspicion of pulmonary infarct. The distinction between a sero-fibrinous and a purulent effusion can only be made with certainty by puncture; no other method is at all reliable (see EMPYEMA). In a child an effusion is much more likely to be purulent than in an adult, and in a pneumococcal case the general symptoms may be quite slight and in no way distinctive or even suggestive of pus.

Prognosis.—Simple pleurisy is rarely fatal unless it occurs as a terminal or secondary infection, as in cases of Bright's disease; but with a large effusion and much displacement of the heart there is always some risk of sudden death from syncope, especially in old people. The sequelæ, however, are frequently important. Extensive adhesions may be left, and, the lung failing to re-expand completely, fibrous changes occur in the collapsed portion, and sometimes bronchiectasis follows. Even in less serious cases the adhesions may hamper the respiratory movements, so that the secretion in the bronchial tubes of the affected area of lung is not properly expelled, and a permanent catarrhal condition, which often forms a starting-point for chronic bronchitis, is set up. It is very common to find at the base of one lung some impaired resonance with weak breath-sounds and rather fine moist crepitations (conveniently known as "adhesion râles"), varying in number from time to time, but never entirely disappearing. In such cases the presence of old pleural adhesions and thickening may confidently be suspected, even though no history of an attack of pleurisy can be obtained. On the other hand, it not infrequently happens that fairly extensive adhesions give rise to no recognizable consequences of any kind.

Most important of all, however, is the fact that a pleuritic attack, no matter how slight, is presumptive evidence of tubercle, unless some other obvious cause is present. In many cases further disease never develops, but the risk, especially of future tuberculosis of the lung, must never be lost sight of, particularly if the patient should be subjected to unfavourable conditions of life. So long as the disease is confined to the pleura the prognosis is fairly good, but once the lung is affected it becomes much more serious, especially as the

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case is then so likely to become one of open tuberculosis, with all the consequent risks.

Treatment.—The general treatment of a case of acute pleurisy is that of any febrile illness of moderate severity. The patient should be kept in bed so long as the temperature is above normal, the bowels should be well opened at the commencement of treatment, and the diet should be regulated by the height of the fever and the appetite; there is no need to withhold solid food if the patient feels able to take it. In view of the probably tuberculous nature of the disease the supply of fresh air should be abundant, but during the early acute pyrexial stage treatment in the open air is not called for, except in hot weather. If there be much fever a simple diuretic and diaphoretic mixture of solution of ammonium acetate and citrate of potash, or an effervescent mixture, may be prescribed, rather perhaps as a solace to the mind of the patient and his friends than for any definite therapeutic effect which it is likely to produce.

The symptom which generally calls for active treatment at the outset is *pain*. In slight cases of secondary pleurisy, such as those which are so common during the course of ordinary phthisis, a simple and effective remedy is firm strapping of the affected side, precisely as for fractured ribs; perhaps also the resultant immobility of the side tends to check effusion. In other cases, sharp counter-irritation by a turpentine fomentation or by a mustard leaf or plaster may give relief. For very severe pain an ice-bag may be exceedingly useful, but must not be used for feeble patients or at the extremes of life. In the worst cases of all, especially in some instances of traumatic pleurisy, the application of three to six leeches may be more effective than anything else and afford almost immediate relief. Occasionally it may be desirable to give a dose of morphia hypodermically. A troublesome, distressing, and ineffective *cough* calls for the administration of sedatives—demulcents, heroin, codeina, or minute doses of morphia in linctus or lozenge.

When *effusion* forms, treatment on somewhat different lines is called for, although it must always be doubtful as to how far we can diminish the exudation of the fluid or hasten its absorption by medical measures, for its amount must depend upon the extent and severity of the pleural inflammation.

Internally we may give a combination of quinine and iodide of potassium:

Ry Quin. hydrochl. gr. ii.
Pot. iod. gr. v.
Syr. aurant. ʒss.
Aq. ad ʒi.

Three or four times a day.

Locally, iodine—equal parts of the strong and weak tinctures—may be painted over the affected side every night until the skin begins to get sore. More effective perhaps are small blisters, one about the size of a penny applied on three or four consecutive nights to different parts of the side. Some prefer to use flying blisters; that is, a blister left on for an hour or so, not long enough to produce definite vesication, and then moved on to another spot.

Whenever an effusion reaches above the angle of the scapula the question of its removal by *paracentesis* arises. As a rule, it is gradually absorbed as soon as the causal inflammation has subsided, but there certainly are cases in which it will remain indefinitely unless drawn off. The tendency, perhaps, is to be unnecessarily anxious to remove an effusion rather than to wait for its probable spontaneous disappearance; active intervention is generally more attractive than mere passive expectancy, both to doctor and patient. It is not possible to formulate precise rules as to when paracentesis should be performed, but the following may be regarded as general indications: (1) Whenever the effusion reaches as high as the third rib, for any further increase will probably give rise to positive intrapleural tension, which involves a very injurious degree of pressure upon the lung and some risk of sudden and fatal syncope. (2) If a smaller effusion remain stationary for a week or more, especially if fever and other indications of active pleural inflammation have subsided. (3) If, even when the effusion is not large, perhaps scarcely above the angle of the scapula, the patient is cyanosed or suffering from much dyspnoea, e.g. in cases in which the opposite lung is diseased. It is not desirable, if it can be avoided, to tap while the temperature remains high, for so long as acute pleurisy is still present the inflammatory exudation will probably rapidly reaccumulate. Fever, however, is no contraindication to the removal of a really large effusion. It has long been a much debated question whether it is desirable to perform paracentesis when the

lung on the affected side is known to be actively tuberculous, for the effusion may act as a natural splint, keeping the lung at rest and so checking the progress of the disease in it. If there be any urgent indication for withdrawal of the fluid, such as severe dyspnoea, this should be done without hesitation; but, seeing that induction of an artificial pneumothorax is now a well-recognized procedure for arresting the progress of a one-sided pulmonary tuberculosis, it would seem very reasonable not to be in a hurry to remove a pleural effusion of moderate size, which keeps the lung at rest in much the same fashion as would a corresponding volume of gas.

Technique of paracentesis.—Removal of fluid from the chest may be effected either by simple

basin; a flow of liquid from the pleural cavity is thus at once set up.

The apparatus used for aspiration (Fig. 82) must, of course, be sterilized thoroughly before being used, and so must the hands of the operator. The patient should lie in a semi-recumbent position on the healthy side. The best place for the puncture is either just below the angle of the scapula or in the midaxillary line, in the sixth space on the right side and in the sixth or seventh on the left side; in children a space higher may be preferable. The point selected should be carefully percussed to be quite sure that it is absolutely dull. The skin should then be well washed with soap and water and painted over with the weak tincture of iodine. If thought desirable, especially in the case of children, the site of puncture may be rendered anæsthetic by injecting a quarter of a grain of eucaine (less in children) or by a spray of ethyl chloride. The left forefinger should be pressed firmly into the intercostal space selected and the trocar passed in vertically close beside it. In a small child with a thin chest-wall it should not be introduced for more than an inch. After the withdrawal of the cannula, the tiny wound left should be covered with a small piece of gauze and a little collodion.

When exploring the chest with a small needle, care should be taken to prevent the patient making any sudden movement, and if the direction of the needle is altered after its introduction the rib should not be used as a fulcrum, for in either case the needle may break and its end be left in the chest. In performing paracentesis these risks are less, as the cannula is larger and stronger than an exploring needle, but they are not altogether negligible.

The whole of the effusion may, as a rule, safely be withdrawn at one time, if not allowed to escape too quickly, but in the case of an elderly patient with a large collection it may be better to leave some behind, thus diminishing the danger of syncope. The residual fluid often undergoes spontaneous absorption. In any circumstances, the removal should be stopped if the patient complain of severe pain in the side, if he begin to cough violently, if he show signs of faintness, or if the fluid, at first clear, become increasingly bloodstained—a change due, doubtless, to the diminution of pressure upon the engorged vessels on the surface of the inflamed pleura.

The risks of paracentesis are very slight pro-

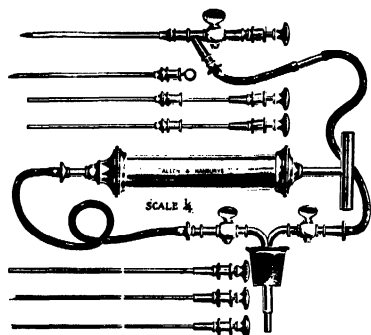


Fig. 82.—Potain's aspirator.

siphon action or by aspiration. For many years the latter was used almost exclusively, but there is no doubt that siphonage is, generally, quite sufficient, and it has the important advantages that the apparatus required is simpler, cheaper, less alarming to the patient, and far less liable to get out of order; it also obviates the risk from too great a suction action being exerted upon the lung. A trocar and cannula similar to those employed for paracentesis abdominis are used. About the middle of the cannula there is a lateral opening or by-way, to which is attached a piece of indiarubber tubing sufficiently long to reach to a basin on the floor containing sterilized water. The establishment of the siphon action is facilitated by first filling the tube with sterilized water and, as soon as the cannula has been inserted into the chest and the trocar withdrawn, lowering the open end into the

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vided that proper antiseptic precautions are taken. Hæmorrhage into the pleural cavity, pneumothorax, subcutaneous emphysema, and faintness or syncope, occasionally fatal, have been recorded. Sometimes an acute œdema of the lungs develops shortly after tapping, with abundant expectoration of watery, frothy, albuminous fluid, and often rapid death from asphyxia. It occurs mostly in patients after middle life, and can neither be foreseen nor prevented. Fortunately it is very rare. For its treatment, atropine should be given hypodermically.

Not uncommonly a second paracentesis is necessary, and very occasionally the fluid re-accumulates repeatedly. In such cases the presence of a new growth should always be suspected, but the condition may occur in ordinary tuberculous pleurisy, especially if the effusion has been left too long, with the result that the lung has been so much compressed that it will not re-expand. In some such instances the pleural cavity has been freely opened and drained, but this should be avoided whenever possible, as an empyema is very likely to result. It is better to persevere with paracentesis and, after each withdrawal, to inject 20 or 30 drops of a 1-in-1,000 solution of adrenalin chloride into the pleural cavity.

In the treatment of the convalescent stage of pleurisy the all-important point to bear in mind is that the condition is in all probability tuberculous, and that the patient should be dealt with, especially in regard to his future life and work, on the same lines as one who has shown signs of early phthisis. Even if the attack has not gone beyond the dry stage and the constitutional symptoms have been quite slight, the disease is still most likely to be tuberculous, and afterwards the same general treatment is called for as in cases of a far more severe type. As has already been stated, the chances of permanent recovery are much better when tuberculous disease is confined to the pleura than when it has attacked the lung itself, and there is no doubt that a large number of patients who have had pleurisy recover completely and permanently without any very prolonged and rigid care subsequently; nevertheless, all such patients should be kept under observation as long as possible, and if convalescence does not proceed satisfactorily, if the general health is not re-established completely, if there should be any indication of a recurrence of the disease, or any obscure pyrexia,

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however slight, full treatment at a sanatorium should be recommended.

It is important to obtain as complete re-expansion of the lung as possible, and, with this object, as soon as the effusion has been nearly absorbed the patient should use Woulfe's bottles (Fig. 83) and take systematic breathing exercises. Later on, graduated exercises, especially on slopes, are useful, and, best of all, the patient should, when practicable, spend several weeks or even months at some Alpine resort, where the rarefied air combined with easy hill-climbing will do more than anything else to expand his lungs and, at the same time, improve his general health. If such a change be impracticable he should, at least, have as

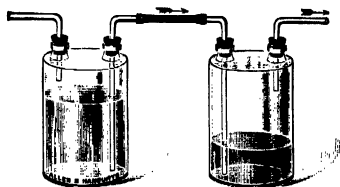


Fig. 83.—Woulfe's bottles.

long a holiday as possible in the open air. Ordinary tonics, together with cod-liver oil and malt, may be given as indicated by the general condition.

J. WALTER CARR.

PLEURISY, DIAPHRAGMATIC.—Occasionally pleurisy affects mainly or even exclusively the pleura covering the upper surface of the diaphragm. This sometimes happens when inflammation extends through the diaphragm as a result of abdominal disease. In this variety of pleurisy the **physical signs** are often very slight; there is usually no friction, and, in the majority of instances, not much effusion, so that there is little or no dullness on percussion. On the other hand, the **symptoms** are often not only exceedingly severe, but may be of a most misleading character. The patient sits up in bed, leaning forward, in order to restrict the movements of the diaphragm and at the same time give thoracic breathing the freest possible play. The respirations are very short and rapid, and associated with intense pain, referred mainly to the line of insertion of the diaphragm, particularly to the epigastric and hypochondriac regions and to the line of the tenth rib. There may also be marked tenderness in the

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same situations. The pain sometimes spreads upwards to the shoulder. Hiccough and a little vomiting may occur. As a rule, there is slight fever. The patient's condition is one of extreme distress, and may raise a very strong suspicion of acute abdominal disease, especially of acute appendicitis should the pleurisy be on the right side. The position which the patient assumes is, however, a very suggestive one. The situation of the tenderness should be noted, and, even though there is no friction, yet the breath-sounds over the base of the lung on the affected side will probably be weaker than on the healthy side.

Prognosis and treatment.—The more acute symptoms generally pass off in a day or two, and the total duration of the illness is usually short, except in those very exceptional cases in which pus forms between the lung and the diaphragm. The treatment does not differ materially from that of ordinary pleurisy; the side should be vigorously counter-irritated by poultices, turpentine, or mustard, and if the pain and dyspnoea are very severe a hypodermic injection of morphia should be given.

J. WALTER CARR.

PLEURODYNIA (see MYALGIA).

PLUMBISM (see Lead Poisoning, under POISONS AND POISONING).

PNEUMONIA.—It is customary to classify pneumonia according to its anatomical disposition and to recognize a lobar and a lobular form (broncho-pneumonia). This arrangement is not very satisfactory and has given rise to considerable difference of opinion as to the relative frequency of the two varieties. The divergence of view has been most evident in the case of pneumonia in children, one school considering that lobar pneumonia is very rare in young children, particularly in those under 2 years of age, the other that, on the contrary, it is relatively common. Primary broncho-pneumonia of children is recognized as being due to the pneumococcus, and runs a course which, for the most part, resembles that of lobar pneumonia. Anatomically, however, the consolidation is seen to be placed around the bronchi and to be patchy. To the pathologist the disease is a broncho-pneumonia, but, despite the patchy and bilateral disposition, the clinician sees more resemblance in it to lobar pneumonia than to the other varieties of broncho-pneumonia. In primary broncho-pneumonia of children, too, the peribronchial

patches of consolidation may be so aggregated together that the whole or the greater part of a lobe may be continuously solid, so that, even anatomically, lobar pneumonia is closely simulated. If it were generally recognized that the clinical features of pneumonia depend, not upon the distribution of the consolidated areas, but upon the nature of the causal organism, the present confusion would disappear. Both schools recognize the frequency of pneumococcal infection of the lung in early life, but apply a different terminology to the pneumonia produced, the one calling it lobar pneumonia from its general resemblance to that disease, the other, more exactly, broncho-pneumonia from its anatomical disposition. For these reasons a bacteriological classification is very desirable. In the present state of our knowledge, however, it is impracticable, and the usual classification is adhered to in this article.

I. LOBAR PNEUMONIA (CROUPOUS OR FIBRINOUS PNEUMONIA)

A general infective disease manifesting itself locally by inflammation of the lungs, and characterized by toxæmia of varying intensity, and by fever which generally ends by crisis.

Incidence.—Pneumonia is a very common and destructive disease. In 1919 the Registrar-General's Report for England and Wales gives the total number of deaths from pneumonia of all forms as 38,949, of which 21,981 were of males and 16,968 of females. In the case of males this corresponds to a death-rate of 127.9 per 100,000 of the population, and in that of females of 86.5 per 100,000.

Many epidemics of pneumonia have been described in schools, gaols, barracks, ships, and hospitals. An epidemic incidence depends upon an enhanced virulence of the pneumococcus, increased by each successive transmission from patient to patient. Liability to epidemic outbreaks is increased if coincident infection by streptococci occurs. A noteworthy example of epidemicity in pneumonia was that which accompanied the influenza outbreaks of 1918-19, in which mixed infections were the rule. Streptococci were often found in the sputum, lungs, spleen, and blood.

Etiology. Age.—Lobar pneumonia occurs at all ages. It has been described in the newly-born and is commonly the immediate cause of death in the aged. Owing to confusion in the nomenclature there is considerable difference of opinion as to the relative frequency of pneu-

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monia and broncho-pneumonia in young children. Pneumococcal infection is certainly common but under 3 years of age the consolidation is generally peribronchial in distribution. Later, the lobar form is encountered more frequently. More persons die from lobar pneumonia between the ages of 35 and 40 than at any other period, but this is no index of the age-incidence of the disease, for its prognosis is relatively good in children. The liability to pneumonia is considerable under 6 years of age, and, according to Osler, diminishes between 6 and 15, to rise subsequently with each decade. G. W. Norris's figures show that 40.2 per cent. of all cases occur between the ages of 10 and 30.

Sex.—Men are more often affected than women in about the proportion of 3 to 2. In children this difference in sex-incidence does not hold good. It has been thought that the greater frequency in men is due to greater exposure in the course of their occupations, but during the War the death-rate among women fell, although their work lay out of doors more than before. A truer explanation is probably to be found in alcoholism.

Geographical.—The disease occurs in all parts of the world, but is most common in the temperate zones. It is met with in tropical countries as well as in those in which the temperature is low; in the former, however, it is commoner at high altitudes, where the fluctuations in temperature are greatest. Dwellers in cities are more liable than those in the country, despite their less exposure to inclement weather; this is probably more than counterbalanced by the crowding and less sanitary conditions.

Seasonal.—The changeable seasons are those in which pneumonia is most prevalent. An influx of cases occurs in the early spring. In the metropolis most cases are seen between the end of March and the end of June (Herringham).

Cold.—There is a notable connexion between pneumonia and exposure to wet and cold, but the nature of this relationship is difficult to understand. After a spell of inclement weather, especially when accompanied by cold and wet winds, many cases of pneumonia are seen. Cold itself does not appear to be a cause, for the coldest months of the year are not those in which pneumonia is most prevalent. A definite history of exposure and wetting is often obtainable, and immersion is an occasional antecedent.

Racial.—All races are not equally susceptible. The Celt would seem to be more liable than the Teuton, and in the United States the mortality among negroes is greater than that among the white population. The Chinese are particularly resistant. In the South African mines pneumonia is very prevalent among the immigrant negroes and causes many deaths. Between August, 1910, and December, 1912, 45,291 tropical natives were recruited for the working of the mines, and of these nearly 40 per thousand developed pneumonia, with a death-rate from this disease of 15.7 per thousand. Pneumonia accounted for 49 per cent. of all deaths. How far the incidence is explained by an absence of immunity, and how far by the occupation, is still to be determined. It is recorded that the Laplanders, when they leave the frozen north and emigrate to a temperate zone, usually die from this disease.

Alcoholism definitely predisposes to pneumonia. In this regard the greater exposure to which drunkards subject themselves must be taken into consideration.

Trauma is an exceptional cause in civil life and accounts for at most 3-4 per cent. of the cases. The injury sustained is usually a blow on the chest.

Inhalation of irritating gases, such as chlorine or ammonia or the gases of war, generally causes a widespread bronchitis or broncho-pneumonia, but occasionally the consolidation is lobar in form. The same is true of anæsthetics. The relation between pneumonia and operations and anæsthetics is considered later.

Occupational.—Certain industrial workers, notably cotton-strippers and workers in basic slag, are especially liable to pneumonia (*see PNEUMONOCOINOSSES*).

Previous attacks.—The immunity conferred by an attack is very short-lived, and it would seem, indeed, that previous attacks favour subsequent ones. Several attacks in one individual are very common, especially in early life.

Acute and chronic illness.—Pneumonia is a frequent complication of influenza and pulmonary tuberculosis, and occurs less frequently in enteric fever, acute rheumatism, and acute nephritis. The disease is prone to affect patients suffering from a chronic enfeebling disorder and to bring it to a fatal termination. It is thus an occasional cause of death in cases of chronic Bright's disease, hepatic cirrhosis, cardio-vascular lesions, diabetes, and hemiplegia and other paralyzing diseases of

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the nervous system. It is one of the chief causes of death in the insane.

Bacteriological considerations.—The pneumococcus (PLATE 4, Fig. 3, Vol. I, facing p. 146) was first recognized as the cause of pneumonia by Fraenkel in 1884. It is a lance-shaped diplococcus, readily stained by the Gram method. Regarded by many as closely allied to the streptococcus pyogenes group, it is capable of being distinguished from them by bacteriological methods. Many varieties have been cultivated, and the morphological and cultural characters are far from constant. Cole, summarizing recent work at the Hospital of the Rockefeller Institute, divides the pneumococci into four groups according to their immunity reactions. Types I and II are spoken of as "fixed types." Though possessing no constant morphological or cultural characters, they show distinctive immunity reactions. Thus, if an animal be inoculated with organisms belonging to Type I, its serum agglutinates all organisms of this type and protects mice against infection by any organism of the same type. The same is true of Type II. Type III comprises organisms of the pneumococcus mucosus group; they are not agglutinable, nor can an immune serum be obtained by inoculation of animals. To Type IV are relegated those strains of pneumococci possessing no common immunological characters. In 500 cases of pneumonia which were studied to determine the relative frequency of infection by the four types, from 60-65 per cent. were shown to be due to pneumococci of Types I and II, 10-15 per cent. to pneumococci of Type III, whilst in the remaining 25 per cent. the pneumococci belonged to Type IV.

Pneumococci are common inhabitants of the mouth and pharynx of healthy persons, but do not long survive outside the body, persisting at most for about ten days, and then only under very favourable conditions. The workers at the Rockefeller Institute have shown that the variety of pneumococcus found most frequently in the mouth is not that which most commonly causes pneumonia. Thus, in 75 per cent. of the instances in which pneumococci were found in the mouths of normal persons, the organism belonged to Type IV. In less than 12 per cent. of instances was the organism of the so-called "fixed" Types I and II, and in 17 per cent. it belonged to Type III. When organisms of Types I and II were found in the mouths of normal persons it was possible to trace a close association

between the person harbouring them and a case of pneumonia due to organisms of the same type; moreover, pneumococci of these types were prone to disappear from the mouth after a short time. It would appear from these investigations that pneumonias due to pneumococci of the "fixed" types are specific infectious diseases, the infection being transmitted from a previous case, either directly or by the intervention of a carrier. In pneumonia due to cocci belonging to Type IV the infection is probably autogenous. The position with regard to organisms of Type III is not clear, for, though highly virulent and widely distributed in the mouths of normal persons, they are only associated with pneumonia, as has been said, in 10 to 15 per cent. of cases.

Other organisms which have been found in association with lobar pneumonia are the pneumobacillus of Friedländer, the Streptococcus pyogenes, Staphylococcus aureus, B. diphtheriae, B. influenzae, B. typhosus, B. coli communis, the gonococcus and putrefactive bacteria. In nearly all instances these organisms merely play the part of secondary infection, and, though modifying the course of the disease, cannot be said to be its cause. Rare examples of pure infection by the pneumobacillus of Friedländer, streptococci, B. typhosus, B. coli communis, and the gonococcus have been described.

Cases in which the pneumonia is due to organisms other than the pneumococcus, or in which secondary infection is prominent, are prone to run an irregular course; complications are more frequent and prognosis is graver.

Pneumococci are readily recovered from the sputum and lung and are generally obtainable from the blood. Rosenow, in a series of cases, found them in the blood in every instance, and in some cases before physical signs were evident, an observation which favours the view that the pulmonary infection occurs by the blood-stream. Though pneumococæmia is perhaps a constant feature of lobar pneumonia, the inflammation of the lungs is generally the only lesion. Plurality of pneumococcal lesions is possibly explained by actual multiplication of the organisms in the blood, as distinct from a simple bacteriæmia. Recently the organism has been grown from the spinal fluid in 14 out of 16 fatal cases by Rohdenburg and van der Beer. On the other hand, among 43 cases which recovered, the fluid yielded negative results in all but 15.

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Morbid anatomy.—Three stages of inflammation are recognized in the lung. In the first, that of *acute congestion or engorgement*, the lung is deep red in colour, still crepitant, though firmer to the touch than normal, and floats in water. It can be inflated by pumping in air through a bronchus. On section the cut surface drips blood and serum. The capillaries and venules are dilated and engorged with blood, and the alveoli contain some red corpuscles and a few alveolar cells and transuded lymph. The cells lining the alveoli are swollen.

The stage of *red hepatization* follows. In it the affected lung or part of the lung is solid and friable, appears larger than normal, and often bears the impress of the ribs. The weight of the lung is increased, and may be as much as three or four times the normal. It sinks in water and cannot now be distended by insufflation. On section the surface is brownish-red in colour and drier than before. The greatly distended alveoli give a granular appearance, more noticeable in adults than in children. Scraping with a knife removes a reddish viscid fluid containing granular masses. Microscopically, the alveoli are found to be distended with red blood-cells, polymorphonuclear cells, and mononuclear phagocytes, and shed alveolar epithelium enmeshed in a fibrin network. The alveolar walls are congested, and the fibrous septa may be swollen and infiltrated with leucocytes. The bronchi are reddened and contain a serous frothy exudate. In the affected area there is bronchitis of the smaller tubes, which may contain plugs or casts of fibrin. The contiguous pleura is always affected if the surface of the lung takes part in the consolidation. There may be only loss of lustre with a thin layer of exudate, or a thick patch of fibrinous lymph or pyolymph. Some degree of serous effusion is the rule. The bronchial lymph-glands are swollen, pulpy, and oedematous. The unaffected part of the lung may be congested or oedematous, or may be normal in appearance.

Resolution may occur at this stage, or the inflammatory process may be continued to the stage of *grey hepatization*, in which the lung remains solid but becomes paler in colour, resembling granite in appearance, though even more friable than before. The cut surface is moister, and a creamy turbid fluid can be scraped off or made to exude by pressure. The alveoli now contain a less defined fibrin network and fewer red cells than before; instead,

the leucocytes are in abundance, many of them showing degenerative changes. The congestion of the vessels is less or absent. The pleural exudate may have become partially organized.

Red and grey hepatization sometimes occur at the same time, one part of the lung being in the stage of red, the other in that of grey hepatization. The two stages may be detected side by side in the same lobe.

The term *purulent infiltration* is sometimes applied to a later stage of grey hepatization in which the lung has become softer and is bathed in creamy pus.

Resolution is brought about by the proteolytic action of enzymes derived from the phagocytes. Some of the exudate is expectorated, except in children, but the greater part is absorbed and is excreted by the kidneys, producing a nitrogen excess in the urine, which has been shown to correspond with the progress of the resolution. In favourable cases the lung returns to its normal condition. In others, local or diffuse suppuration, gangrene, or chronic interstitial pneumonia develops.

Lesions in other organs.—The lesions in other organs may, for the most part, be divided into those which are due to the toxins of the disease and are comparable to the changes found in other infective fevers, and those which are due to pneumococcal invasion. Into the first group fall cloudy swelling and fatty degeneration of the heart, liver, and kidneys, degeneration of nerve-cells, and congestion of the spleen; into the second, empyema, pericarditis, endocarditis, meningitis, peritonitis, arthritis, and otitis media. Croupous gastritis and colitis are sometimes found. The cavities of the heart, especially the right auricle and ventricle, are dilated, and contain much firm adherent clot, which projects for some distance into the great vessels. This clot is generally regarded as occurring post mortem, but Fleming believes that it appears before death.

Symptomatology. Incubation.—The period of incubation is short, probably no more than two or three days, and has been noted as only twenty-four hours during an epidemic.

Onset.—The disease begins abruptly with pain in the side and a rapidly rising temperature, which reaches perhaps 103° or 105° F. in the course of a few hours. An early rigor is frequent in adults. In children vomiting is the rule, sometimes accompanied by diarrhoea, whilst convulsions are relatively uncommon. Other initial symptoms are head

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ache, sore throat, epistaxis, and hæmoptysis. Chilliness or shivering may replace a more definite rigor. In the old and debilitated, in drunkards and the insane, and in those suffering from acute or chronic diseases, the onset is apt to be so insidious as to be unsuspected.

Course.—Following the initial chill and rise of temperature the skin becomes flushed, hot and dry, and the patient restless, complaining of throbbing in the head, headache, general pains, and thirst. The pain in the side is often severe, and made worse by breathing. Respirations are rapid and shallow, painful, accompanied by dilatation of the *alæ nasi* and, in children particularly, by an expiratory grunt. They are interrupted from time to time by a short, painful cough. Herpes frequently develops on the lips. The tongue is white and furred. After the onset the pulse becomes full and bounding, and increased in rate in proportion to the fever, but relatively slower than the respiration. The expression is anxious, the eyes are bright and the pupils often unequal. In a few days a degree of cyanosis is usually seen. The restlessness may merge into delirium, especially at night, and sleep is lost from this and from the pain, cough, and engorgement of the right side of the heart. Constipation is the rule, and appetite is lost. At first expectoration is usually absent, but in the course of a few days blood-stained, tenacious sputum is coughed up with difficulty, to become more fluid and be expelled more easily later. After a variable number of days the temperature drops by crisis, the respirations become easier, and the patient rapidly passes from a condition of pain and distress to one of comfort.

The symptoms considered more in detail.

Fever.—In no disease is the temperature chart more characteristic. The rise is rapid, the fastigium being reached in 10-12 hours. Thereafter the temperature fluctuates very little in a typical case, remaining perhaps in the neighbourhood of 103° or 104° F. until the crisis. With the crisis the temperature falls rapidly to normal or lower, usually within from five to twelve hours. The crisis of pneumonia connotes far more than a sudden defervescence. At the same time breathing becomes easy and slow, pain disappears, the pulse becomes steady, there is usually a profuse sweat, and the patient generally sinks into a deep and refreshing sleep. The crisis marks the end of the toxæmia, and during this period the patient

passes from danger and distress to safety and ease.

Sometimes the temperature falls rapidly by a few degrees, even nearly to the normal line, and then rises again (pseudocrisis); or it may rise a few degrees before the final drop (pre-critical rise). A crisis in two stages is not uncommon, especially in children. When this occurs the temperature falls perhaps to normal or even below, then rises again, possibly even above the former level, to fall again with the end of the fever. In children, in the old and feeble, and in alcoholics the fever may end by lysis, and the same is true of cases of pneumonia in which organisms other than the pneumococcus are present either as the primal cause or as a pronounced secondary infection. When resolution is delayed the fever may persist for several weeks until it falls gradually to normal. In complicated cases, in alcoholics, and at the extremes of life the temperature is apt to be irregular. Its range is often low in profoundly toxic cases, in the aged, and in drunkards; indeed, in exceptional instances no rise occurs at all.

Dyspnoea. The respirations are rapid, often amounting to from 30 to 50 per minute in adults and exceeding 80 in children. These are shallow and painful from the pleurisy, and accompanied by dilatation of the *alæ nasi* and an expiratory grunt. Sometimes thoracic movements are so restrained that the breathing is almost entirely abdominal. A type of respiration is sometimes seen, in children, in which the usual rhythm is reversed, each respiratory act beginning with expiration. The rhythm is thus expiration—inspiration—pause, and both expiration and the pause are associated with muscular action, the chest being held rigid with the glottis closed or partially so. The pulse-respiration ratio is altered, perhaps being 2:1, or even approximating still nearer to unity. The dyspnoea is partly accounted for by the pain and fever and the diminished area of normal lung, but is chiefly due to the toxæmia, for the respiratory rate falls to normal at the crisis, although, by physical signs, the area of solid lung has not diminished. Probably an important factor is the action of toxins upon the respiratory centre.

Cough is almost always present, but may be absent in apical pneumonia, in the aged and those enfeebled by other diseases, in young children, and in cases complicated by delirium tremens. It is short, restrained, and distress-

ing, and causes the patient to groan and hold his side from pain. Sudden cessation of coughing in a patient in whom this symptom has been well marked is an evil omen.

Pain is one of the earliest symptoms, and is due to the accompanying pleurisy, being absent in central pneumonias and absent or inconsiderable when the consolidation is apical. It is generally situated in the axillary or mammary region, and is made worse by deep inspirations and by coughing. Sometimes it is wholly or mainly abdominal, and when the right lower lobe is affected may be referred to the right iliac fossa and suggest appendicitis. Præcordial pain may be distressing if pericarditis supervenes.

Expectoration.—At first little or no sputum is coughed up, though occasionally there is a brisk hæmoptysis. After two or three days the sputum becomes more abundant, but is still difficult to expel. It is mucoid, viscid, and so tenacious as to remain in an inverted spittoon. It contains blood in a varying amount; in most cases this is bright red at first and then becomes rusty, but in some a considerable amount of bright-red blood is expectorated throughout the illness. In others, streaks of bright blood represent the only hæmorrhage. Later the sputum is muco-purulent and is generally brought up with more ease and in greater quantity with the onset of resolution. In children and sometimes in old people there is no expectoration.

Appearance.—The patient generally lies more comfortably on the affected side and in a semi-recumbent position; the face is more flushed on the same side. The lips are often cyanotic after the first few days and sometimes from the beginning. Herpes is more frequent than in any other affection, and may appear on either or on both sides of the lips, or on the alæ or septum of the nose, but is rather more common on the side of the consolidated lung. These features, with the rapid, restrained, and perhaps grunting respirations, the rigid outline of the accessory respiratory muscles, and the rhythmically dilating nostrils, present a picture which is very characteristic.

Physical signs.—During the stage of congestion breath-sounds are weak over the affected area, and fine sharp crepitations (hair-like crepitations) become audible. The percussion note may be impaired or may be higher pitched than normal, or skodaic in quality. Pleural friction can sometimes be heard and felt.

With the advent of consolidation its characteristic signs make their appearance: deficient movement, dullness, bronchial breathing, bronchophony, and increased vocal fremitus. With consolidation of a lower lobe the movement over the apex may appear exaggerated. The dullness, though definite, is not so wooden or resistant as with fluid. Over the lung bordering on the solid area a tympanitic note may be obtained. The bronchial breathing is usually more definite than in any other condition, and incorporated with it is a hard "ch" sound; in other cases it is softer and blowing in quality. Sometimes the exaggerated vocal resonance has an echoing nasal sound (ægophony), but this is seldom so pronounced as in pleural effusion. The measurement of the chest on the affected side may exceed that of the other.

Resolution is denoted by the advent of many sharp clicking râles (subcrepitant râles, *redux crepitations*) over the solid area, and a gradual diminution of the dullness and other signs of consolidation, with coincidental return of normal breath-sounds. Tympanitic or skodaic resonance may appear during the period of resolution.

Under certain conditions the usual physical signs are modified or lacking. Thus, in "massive pneumonia," in which the air-passages are plugged with fibrin, breath-sounds are inaudible and vocal resonance and fremitus may be diminished or lost. In central pneumonia the chest signs may be very ill-defined or absent, or may appear for the first time after the crisis.

Two precautions should be observed when eliciting the physical signs. Percussion should be very gently applied, more especially in children, not only for humanitarian reasons but also because vigorous percussion elicits the masking drum-like note of the mattress below. Secondly, no reliance should be placed upon the signs obtained over the side of the chest on which the patient is lying. Whenever possible the patient should be turned over, that each side can be examined uncompressed. It is well also to remember that the auscultatory signs, especially the bronchial breathing, may be transmitted to the other lung. This is particularly noticeable in children.

Distribution of the consolidation.—In 100 autopsies Sir W. Osler found that the right lung was affected in 57 cases, the left in 32, and both in 15. In 27 cases practically the whole lung was consolidated. In 34 the lower lobe alone was affected, and in 13 the

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consolidation was confined to the upper lobe. When double, the lower lobes were usually affected together, but in three instances the lower lobe of one lung and the upper lobe of the other were attacked. In three cases, too, both upper lobes were solid.

Circulatory system. *The pulse.*—At the onset and during the initial chill the pulse may be small and feeble, but during the time when the fever is at its height is full and bounding. It is increased in rate approximately in proportion to the fever, but to a less extent than the respirations. In severe cases, when the toxæmia is profound or the resistance low, as in alcoholics and old people, the pulse may be rapid and weak from the onset. Some loss of strength and increase in rate are usually detected before the crisis and during its course, but there are cases in which the pulse remains full and strong throughout.

The heart.—Owing to interference with the circulation through the lungs, a degree of dilatation of the right ventricle is usually present; in some cases, especially in plethoric subjects and in those with extensive consolidation, this may be considerable and associated with the usual evidences of right-heart failure—cyanosis, dilatation of the neck veins, increased cardiac dullness to the right and enlargement and tenderness of the liver being present, and, less frequently, oedema of the legs. This mechanical result is of less importance than those produced by the toxæmia. The toxins of pneumonia reduce the muscular efficiency of the heart and are capable of causing degeneration of the myocardium, so that the beat in severely toxic cases becomes feeble and quick, perhaps irregular, and the output of blood is diminished. In these patients the face and lips are often pale with a cyanotic tinge, or are ashy-grey. Collapse may occur, especially near the crisis, the heart-beat becoming very feeble and its sounds similar and spaced, the pulse small and soft, the extremities cold and blue, and the skin clammy—an attack which may be fatal. Cardiac failure is the usual cause of death in pneumonia, but the effect of the toxins upon the medullary centres plays at least as important a rôle in the production of collapse as the degeneration of the myocardium. By it vagal control is cut off and the heart increases its beats and may run itself to death. Some left ventricular dilatation is often detectable, and systolic murmurs at the apex and base are often heard. The pulmonary second

sound is accentuated as a rule, but is weak or may be lost if heart failure supervenes.

The *blood-pressure* is unaltered at first, and usually well maintained. A gradually falling pressure indicates increasing cardiac weakness and is a valuable indication of the need for stimulation. G. A. Gibson has drawn attention to the blood-pressure as a guide to prognosis. If the blood-pressure as registered in millimetres of Hg falls below the pulse-rate the outlook is bad. Sudden falls do not occur apart from collapse.

Blood.—The most important change is polymorphonuclear leucocytosis, which follows approximately the same curve as the fever, but may continue longer, especially if resolution is also delayed. The leucocytes are generally more than doubled in number and may attain 100,000 per c.mm. No increase or an actual leucopenia occurs in severely toxic cases, and is of grave omen. The blood-platelets and fibrin elements are greatly increased. The carbon-dioxide content is nearly always diminished.

Nervous symptoms.—Headache is often complained of from the onset. Some degree of delirium, often with hallucinations, is common during the height of the fever, especially at night. Occasionally the patient becomes maniacal, and may need careful watching to prevent him from self-injury or suicide. In such cases, and in alcoholics, in whom delirium tremens is a common complication, the pneumonia is easily overlooked. In toxic cases the patient is dull and lethargic, and may rapidly sink into stupor with low muttering delirium. Mental symptoms may persist after the crisis, and delusions may remain for several weeks, but ultimate recovery is the rule. In children an attack of pneumonia may closely simulate meningitis and begin with vomiting, pain in the head, head retraction, rigidity of the neck, great irritability, and perhaps convulsions (meningismus).

Urine.—The urine is diminished in amount during the fever, of high specific gravity, turbid from urates, and highly coloured from excess of pigment. A trace of albumin is common. Nitrogen is in excess and falls to normal soon after the crisis, except in cases of delayed resolution, when it remains high. Chlorides, sodium, and calcium are all retained in pneumonia. The reduction of the chlorides in the urine is of diagnostic value.

Laryngitis is not uncommon in the early stages and may precede the onset.

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Complications.—*Pleurisy* is so constant a feature of lobar pneumonia that it can hardly be regarded as a complication. It varies from a small patch of lustreless and congested pleura to a felt-like mass of fibrinous lymph or pyo-lymph over the whole or the greater part of the solid lung.

Pleuritic friction may be audible at the onset of the disease, throughout its course, or only in the later stages; it may not be heard at all. Comparatively rarely is there a collection of serous fluid of sufficient amount to be recognized clinically.

Occasionally pleurisy occurs on the side opposite to that of the pneumonia. Diaphragmatic pleurisy is a very painful complication, and is serious from the exhaustion caused by loss of sleep.

Empyema is more common than recognizable serous effusion and occurs in from 2 to 3 per cent. of all cases. It is found generally on the same side as the consolidation, and represents an invasion of the pleura by infection from the lung. In these circumstances it is discovered towards the end of the pneumonia, and is often suspected first because the temperature rises again within a few days after the crisis, and the dullness instead of clearing up becomes enhanced. The breath-sounds and vocal resonance are distant or lost in adults, but in children bronchial breathing and voice-sounds often remain, or even become exaggerated. Displacement of the heart to the opposite side is the diagnostic sign of greatest value.

In cases of generalized infection, empyema may occur early in the disease and may affect either or both pleuræ. In these serious cases death often anticipates any considerable collection of pus, and the condition is therefore first detected post mortem. If, with persistence or recurrence of fever, the dullness deepens or more resistance is met with, exploratory puncture should be performed.

Pulmonary complications.—*Delayed resolution*, follows in from 3 to 4 per cent. of cases. Crisis may occur in the usual way and the patient lose all serious symptoms, but the local signs fail to clear up. The dullness, bronchial breathing, and moist sounds continue for several days or weeks, perhaps finally to disappear completely, even after persisting for as long as three months. In other cases the temperature falls slowly by lysis, but either fails to reach the normal line or, having done so, rises slightly again, to continue irregularly

perhaps for weeks. Persistence of the physical signs accompanies the slight fever, and the patient fails to convalesce. Ultimately signs and symptoms gradually disappear. In yet other cases with persistent physical signs the fever returns after a period of apyrexia. Delayed resolution is more properly designated "continued infection." The virulence has diminished, but the inflammation continues, and interstitial pneumonia and consequent fibrosis of the lungs not infrequently follow.

Pulmonary abscess, if one excludes the diffuse suppuration found in the later stages of grey hepatization, is uncommon. A limiting membrane is seldom seen, for when abscesses occur they are usually small and surrounded by necrotic lung. Empyema is generally suspected, and only by operation is it discovered that the pus lies within the lung.

Gangrene of the lung is also only occasional. It is met with especially among feeble elderly patients, alcoholics, and the subjects of diabetes, and is almost invariably fatal.

Chronic pneumonia.—Instead of resolution the inflammatory products may become organized, vascularization of the alveolar contents being followed by fibrous overgrowth from the alveolar walls which themselves become thickened. The fibrosis leads to atrophy of the aerating tissue and to shrinking of the lung, and by traction may cause bronchiectasis.

Cardio-vascular complications.—*Dilatation of the right side of the heart, myocardial degeneration, and collapse* have already been referred to. *Cardiac arrhythmia* is not uncommon; it is usually of the "sinus" variety and is influenced by the respirations. Premature beats are sometimes met with, and heart-block has been noted.

Pericarditis is one of the most important and serious complications, and occurs in slightly more than 1 per cent. of cases. It is usually associated with consolidation on the right side; sometimes also it appears to be related to consolidation of the lingual lobe. It is very insidious in its onset and course, and is usually overlooked despite repeated examinations, the friction sound being often transient or absent. Increasing pallor and distress, with a rapid pulse and pain in the præcordium, orthopnoea, and persistent fever, should arouse suspicion of pericarditis, especially when the pulmonary signs appear to indicate a subsidence of the pneumonia. Pericarditis is generally due to infection by contiguity, and

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is almost always accompanied by pleurisy or empyema. Occasionally *mediastinitis* is also apparent in these cases and localized collections of pus are found in the mediastinum; tenderness and dullness and perhaps œdema, to the right of the manubrium and upper part of the gladiolus, are the usual evidences of this rare complication. Generalized infection accounts for the pericarditis in other cases. Pneumonia complicated by pericarditis is nearly always fatal.

Endocarditis was recognized in 144 of 32,349 collected cases, or 0.44 per cent., according to G. W. Norris. At autopsy it was found in 157 of 2,639 cases, or 5.8 per cent. This wide discrepancy between the number of cases detected during life and found post mortem exemplifies the difficulty of diagnosis. Cardiac dilatation and bruits are common in pneumonia, and nothing may be detected in the heart to suggest a new lesion, though the supervention of infarcts in spleen, kidney, brain, or lung may provide sufficient evidence. Endocarditis is twice as common in women as in men, and is ulcerative in about 75 per cent. of the cases. It has been pointed out that though it more often affects the left side of the heart than the right, the proportion of right-sided lesions is greater than in other forms of infective endocarditis. Previous valvular disease is a predisposing factor. Since the lesion is situated in the blood-stream, it is often associated with multiple pneumo-occal lesions and especially with meningitis.

The frequency of *ante-mortem* clotting within the heart is undecided. The tenacious thrombi found post mortem are generally considered to have occurred immediately before or after death, but some hold that their formation is earlier. *Venous thrombosis* is a very occasional sequel in pneumonia, and usually affects the femoral vein.

Gastro-intestinal complications. Vomiting may occur as the result of gastritis or meningitis, may follow severe fits of coughing or be toxic in origin. It is a common initial symptom in children. *Croupous gastritis* and *colitis* are rare complications; with the latter there may be severe diarrhoea. A variety of hæmorrhagic and ulcerative gastritis has been described (Dieulafoy). Less uncommon is *acute dilatation of the stomach*, which may occur early and lead to severe pain in the epigastrium or left hypochondrium; it may cause a fatal collapse. *Meteorism* is not infrequent, though it is less common and troublesome than in

typhoid fever. It is generally toxic in origin, but may be due to colitis or peritonitis.

Peritonitis is rare, and is usually met with in children. It may be general or localized; the former is usually fatal, the latter is serious though more hopeful. *Jaundice* in a mild degree is not very rare. Only occasionally is it at all prominent, and is then of serious import and accompanied by a high mortality. It is generally toxæmic, without alteration in the colour of the stools. Rarely it is associated with cholecystitis and cholangitis.

Nervous complications.—Delirium, mania, stupor, delusions, and meningismus have already been considered (p. 566). Most important is *delirium tremens*, which not only masks the diagnosis but often determines a fatal issue. *Meningitis* is a very fatal result of generalized infection; it is found in 3.4 per cent. of fatal cases and is seldom diagnosed during life, for it is generally vertical and hard to distinguish from the meningeal symptoms of toxæmia. When it supervenes late in the attack or is accompanied by cranial nerve paralysis it is more easily recognized. *Neuritis* is a rare complication. *Hemiplegia* has been described, and may be toxic or due to embolism; *aphasia* may accompany it or occur independently. Other complications are *otitis media*, *parotitis*, *nephritis*, and *arthritis*. The arthritis may be confined to one joint or affect many, and may be simple or suppurative. It may arise during any phase of the pneumonia, or may precede or follow it.

Relapse is a very unusual occurrence in lobar pneumonia. Instances of return of the fever after an apyrexial period are more common, but they are more properly examples of delayed resolution, for the physical signs in the lungs have not cleared up with the cessation of the fever.

Clinical varieties. Pneumonia in children.—The onset is often marked by vomiting, less often by convulsions, and seldom by a definite rigor. During the pyrexia the breathing is more frequently inverted than in adults, and grunting expiration is more usual. The fever may run a typical course, but is more frequently irregular, and more often ends in two stages, a false crisis preceding the true one. Lysis is a more usual mode of termination than in adults. When a crisis occurs it is less abrupt and less exhausting. Children rarely expectorate. Examination of a child with pneumonia may provoke screaming and general tremor, symptoms which are due to delirium

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and hallucinations, but are sometimes erroneously ascribed to "temper." The physical signs in the lungs are often more delayed or ill-defined in children, but in such cases the symptoms alone generally suffice to establish a correct diagnosis. When bronchial breathing and bronchophony are well marked one must expect to hear them over both sides of the chest, so readily are they transmitted. A diagnosis of bilateral consolidation, however, is not justified unless the auscultatory signs are corroborated by the presence of dullness. Pneumonia in children is prone to assume the "cerebral" form, in which meningitis is closely simulated. Successive involvement of different lobes (migratory pneumonia) is met with more commonly than in adults.

The child's heart withstands the toxins of pneumonia better than does that of the adult, and general cardiac failure or distension of the right side of the heart seldom gives cause for alarm. Multiplication of pneumococcal lesions remote from the lung is more common.

Pneumonia in old people.—In the elderly, pneumonia is easily overlooked, for the physical signs are liable to be varying and indistinct and the fever slight, irregular, or even absent. The local and general reaction to the infection is poor, and the patient is prone to succumb to the toxæmia without manifesting the characteristic features of the disease.

Secondary pneumonia is a term which has different applications. As used by some it connotes ordinary pneumococcal pneumonia occurring during the course of other diseases, by others an inflammation of the lung due to specific organisms other than the pneumococcus is meant. Although pneumonia as a complicating disease may run its usual course and be easy of detection, generally speaking it is less distinctive than when it occurs alone, for not only are the symptoms obscured by those of the associated disease, but the course is apt to be irregular and the signs indistinct. It is more often lobular than lobar in distribution, though by consolidation of contiguous lobules lobar pneumonia may be simulated in lobular cases. The chief or only organism in the lung may be the pneumococcus, but often it plays a subsidiary rôle. Friedländer's bacillus, staphylococci, streptococci, the influenza bacillus, Micrococcus catarrhalis, and *B. coli communis* are among the organisms met with in company with the pneumococcus or independently.

In **typhoid fever** the pneumonia may occur early or late in the disease. The case when

first seen may appear to be one of uncomplicated pneumonia, the abdominal symptoms of typhoid fever becoming evident only after some days have elapsed. Persistence of the fever, want of resolution in the lung, and increasing drowsiness of the patient, together with such characteristic signs as enlargement of the spleen and the eruption, then lead to a right diagnosis. When the pneumonia is deferred until the end of the second or third week it obscures the picture less, but itself is often overlooked. There may be little alteration in the respiratory rate, no expectoration, no appreciable change in the temperature chart, whilst the signs in the lungs may readily be mistaken for those of hypostatic congestion or œdema. The pneumococcus is almost invariably present, though often associated with septic organisms. Pure cultures of *B. typhosus* have been obtained.

In **rheumatic fever**, in children, pneumonia is sometimes met with; it differs little from primary pneumonia, but may be patchy in distribution. With rheumatic pericarditis, dullness, bronchial breathing, and increased vocal resonance are often elicited at the left pulmonary base or in the lower part of the inter-scapular region on the left side. Though ascribed by many to pressure from pericardial effusion, the signs certainly indicate a true pneumonia in many instances, and the pneumococcus is the usual organism found. Unless examined for, this variety of intercurrent pneumonia is easily missed, for its symptoms blend with those of the pericarditis. Whether a true rheumatic pneumonia exists is uncertain.

Concerning the relationship between **malaria** and pneumonia there has been great difference of opinion. During epidemics of malaria, pneumonia is recognized as a rare complication, but there is no satisfactory evidence that the malarial parasite is the cause. Cases are seen in which the pneumonia appears to improve *pari passu* with the disappearance of parasites from the blood as the result of treatment by quinine. This was noted occasionally in the soldiers at Salonika.

Influenzal pneumonia is considered under INFLUENZA.

Terminal pneumonia.—In the feeble and debilitated, in Bright's disease, arterio-sclerosis, chronic valvular disease, tuberculosis, diabetes, and cancer, pneumonia is a frequent precursor of death. In these circumstances it is insidious in its onset and course, provokes

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little reaction, and is often discovered for the first time after death. There may be little or no rise of temperature; on account of its shallowness the breathing does not compel attention, though it is usually increased; and the physical signs are perhaps not examined for because of the patient's precarious state. Expectoration may be lacking. Even when a careful examination is made of the chest, nothing distinctive may be found.

Toxic pneumonia (asthenic pneumonia, typhoid pneumonia).—When the infection is very virulent or the patient unduly susceptible, he may be overwhelmed by the toxæmia. There is little or no fever, and no leucocytosis, while the pulse soon becomes rapid, feeble, and perhaps irregular. Mental symptoms are prominent; increasing drowsiness deepens to stupor accompanied by subsultus tendinum and a low muttering delirium. The aspect is dusky grey or cyanotic. Jaundice, diarrhoea, and abdominal distension may occur. The prostration, abdominal symptoms, and nervous phenomena produce a simulation of typhoid fever, and a poverty of physical signs in the chest still further obscures the diagnosis.

Larval pneumonia is the name given to mild and abortive pneumonia in which, after a few characteristic symptoms and a short illness, the disease comes to an end, perhaps within a few days of the onset.

Postanæsthetic and postoperative pneumonia.—Various forms and degrees of consolidation of the lung are met with after operations, whether an anæsthetic be used or not, and after both chloroform and ether anæsthesia. The commonest of these is due to inhalation of organisms from the buccal cavity and nasopharynx, though probably assisted by the irritant effect of the anæsthetic. It is more often lobular than lobar in distribution.

W. Pasteur has analysed the pulmonary complications following 3,559 abdominal operations at the Middlesex Hospital, and found that pneumonia occurred in 88 cases, of which 31 ended fatally. The earliest signs generally made their appearance within forty-eight hours of the operation. Graves's analysis of 2,000 cases shows that there is no constant relationship between the pneumonia and the method and length of the anæsthesia or the degree of shock. It occurs most commonly in the cold months. In his opinion a pre-existing focus in the lung is lighted up by the anæsthesia. The supine position, especially in old patients or in those with a failing heart or too feeble

to cough or breathe deeply, favours hypostatic pneumonia.

Another form of consolidation is that which follows embolism due to thrombosis brought about by the operation. The thrombosis may occur in a vein at the site of the operation or more remotely, though sepsis is accountable for some cases; in others, stagnation of the blood in dilated vessels appears to be the true explanation. Pelvic operations are particularly liable to be followed by pulmonary embolism, but it may succeed a normal labour.

Finally, postoperative consolidation may be due to massive collapse, to which attention has been drawn by W. Pasteur. Many of the so-called postoperative or postanæsthetic pneumonias are really examples of this condition, which closely simulates inflammatory consolidation. It may affect either or both lungs, and sometimes precedes an actual pneumonia. It is most commonly found after operations on the upper part of the abdomen, and is ascribed by Pasteur to deficient movement of the diaphragm. When it is unilateral the heart is displaced towards the collapsed side, the measurement of which is less than that of the normal.

Central pneumonia and massive pneumonia have already been referred to (p. 565).

Diagnosis.—A frank lobar pneumonia is not difficult to diagnose. The sudden onset with pain in the side, rigor, and rapid rise of temperature, the quick respirations, the continuous fever ending by crisis, the appearance of the patient, and the pulmonary signs of consolidation, present a clinical picture which is sufficiently characteristic. In the aged and feeble, in alcoholics, and in the insane, and when pneumonia occurs as an intercurrent disease, more difficulty is encountered, for its advent is more insidious, its course more irregular, and its fever less characteristic, while the physical signs in the lungs are less distinctive. The same is true of very toxic cases. In these the cerebral symptoms of the toxæmia may dominate the picture and quite overshadow the respiratory; while the patient's reaction is so slight that neither fever nor leucocytosis may result.

Pleurisy.—At the onset pleurisy alone may be suspected, especially when pleuritic friction is a prominent feature, but in pleurisy the initial symptoms are generally less severe, rigor is uncommon, and the temperature range is lower. The appearance of consolidation dispels the doubt. Pleural effusion is seldom

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confused with pneumonia in adults. The signs of fluid in the one case and of consolidation in the other lead to a correct diagnosis. In children pleural effusion more closely resembles pneumonia, for bronchial breathing, increased vocal resonance, increase of whispering sounds, and even mucous râles may be heard over the area of the effusion. Greater help is afforded by the dullness and resistance, by the absence of vocal fremitus, and particularly by the position of the heart's apex beat. Fluid is more dull and more resistant, vocal fremitus over it is diminished or lost, and the heart is displaced. Well-marked ægophony is seldom heard above consolidation. Exploratory puncture should be done if any doubt remains, and should not be deferred if there is any suspicion of suppuration.

Appendicitis.—In pneumonia of the right base the pain is often referred to the right side of the abdomen and sometimes to the right iliac fossa. There may be tenderness and rigidity in this region, and if the chest is not examined a diagnosis of appendicitis is readily made. Inadequate examination has led to a mistaken operation in many such cases.

Delirium tremens.—When a patient is saturated with alcohol, delirium tremens may occur so early that the pneumonia is unsuspected; a history of chill or a rise of temperature, however slight, should arouse suspicion, but the only safe procedure is a methodical examination of the lungs in all cases of *mania a potu*.

Meningitis.—The cerebral form of pneumonia, more usual in children than in adults, may resemble meningitis very closely. Headache, screaming, rigidity of the neck, head retraction, rigidity of the limbs, vomiting, photophobia, and fever are common to both. The abruptness of the onset suggests either cerebro-spinal fever, pneumococcal meningitis, or septic meningitis rather than the tuberculous variety. Retention of the flexor reflexes, absence of a rash or of suppurative otitis or other local suppuration, absence of blindness or ocular changes, and the revealing of signs of consolidation by examination of the chest may prevent a wrong diagnosis. It must not be forgotten that pneumococcal meningitis is an occasional complication of pneumonia, and that after death from pneumococcal meningitis some degree of consolidation of the lung is usually found. Pneumonia, too, is numbered among the complications of cerebro-spinal fever. If cerebro-spinal fever is suspected,

lumbar puncture should be performed to establish the diagnosis, for delay in the treatment of cerebro-spinal fever is more fraught with danger than is the performance of the puncture in a case of pneumonia.

Acute pulmonary tuberculosis may at first be indistinguishable from pneumonia. The onset may be abrupt, the temperature high, the dyspnoea urgent, whilst signs of consolidation may be found over the whole of one lung or lobe. Such a case is almost certain to be regarded as pneumonia during the first few days. Distinguishing features are the deeper cyanosis, greater remission in the fever, a more purulent expectoration, and rapid wasting. Detection of choroidal tubercles by ophthalmic examination and discovery of tubercle bacilli in the sputum may settle the diagnosis. Sometimes, especially in children from whom no sputum is obtainable, a correct diagnosis is deferred for a week or more. Then the absence of a crisis or of signs of resolution, together with numerous moist sounds in the lungs, and perhaps signs of excavation, leave no doubt as to the tuberculous nature of the lesion.

Typhoid fever.—The difference in onset and in the localization of the symptoms, together with the dyspnoea, is sufficient to distinguish the two diseases in ordinary cases. Typhoid fever may, however, start abruptly with pneumonia, while toxic pneumonia may simulate very closely the typhoid state. If a patient thought to be suffering from typhoid fever does not lie supine, the diagnosis is open to grave suspicion. Facial herpes is rare in typhoid fever but common in pneumonia. The most valuable means of distinguishing typhoid fever are the rash, the large spleen, and the isolation of the organism from the blood. The Widal reaction in an uninoculated patient is also diagnostic, but its usefulness is marred by the fact that it is unobtainable until the ninth or tenth day.

The diagnosis from *broncho-pneumonia* is considered later (p. 583).

Prognosis.—Pneumonia is one of the chief causes of death, commoner than pulmonary tuberculosis and exceeded in the Registrar-General's Reports only by tuberculosis of all forms. It must be acknowledged, however, that were accurate returns of deaths from syphilis obtainable and were all the deaths from that disease, immediate and remote, classified under one heading, it would probably be found to take the first place.

The case-mortality in the hospital class of

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patients is higher than that seen in private practice. In the former it is approximately about 20 per cent. for all ages, whilst in private practice it is more nearly 15 per cent. Wells collected 223,730 cases which gave a mortality of 18.1 per cent. Among 7,868 cases in the London general hospitals during ten years the mortality was 21 per cent. A collective Investigation Committee found that in 1,065 cases in London the mortality was 1 in 5.5, but when cases associated with intemperance, mental and physical disorders, infectious diseases, and old age were included it appeared to be approximately 1 in 8.

As in other infective diseases, the prognosis depends essentially upon two factors—the degree of virulence of the invading organism and the powers of resistance of the patient. It would appear that certain varieties of the pneumococcus are more baneful than others. Thus Cole and his collaborators at the Rockefeller Institute record the mortality from infection by pneumococci of Type I as 25 per cent., of Type II as 32 per cent., of Type III as 47 per cent., and of Type IV as 6 per cent. According to Sisson and Thompson, infection by the bacillus of Friedländer is almost invariably fatal. There is little doubt that pneumonia due exclusively to the pneumococcus is less serious than that in which other bacteria play a prominent part. When streptococci or other organisms are superadded, or are responsible for the pneumonia, the outlook is more grave. When, again, the virulence is enhanced by transmission from patient to patient, as occurs in epidemics, a high mortality-rate is likely to be met with.

Secondary pneumonia is fraught with more danger than the primary form, for not only is the infection generally a mixed one but also the patient's resistance is already weakened by the antecedent disease.

Several factors in addition must be reviewed in attempting the prognosis of a particular case.

1. **Age.**—Pneumonia is most fatal at the extremes of life. Difference of opinion as to the relative frequency of lobar and lobular pneumonia in children under 2 years of age makes the figures very fallacious, but the outlook in cases of pneumonia at this age is certainly worse than in succeeding years. In the aged, pneumonia may almost be said to be the usual cause of death. At all ages after infancy until 20 the prognosis is uniformly good, the mortality-rate not being more than

3 or 4 per cent. From 20 onwards it gradually increases, being about 20 per cent. between 20 and 30, 30 per cent. between 30 and 40, and 50 per cent. after 60. After the seventh decade it rises abruptly, and thereafter pneumonia is seldom recovered from.

2. **Previous condition of the patient.**—Patients already enfeebled by poor food, a weakly constitution, overwork, or disease withstand pneumonia badly. In this connexion *alcoholism* stands out as of first-rate importance, and accounts for the majority of deaths from pneumonia in robust-looking individuals in the prime of life, especially those whose tissues have had to withstand the wear and tear of heavy manual labour. Such degenerative changes as arterio-sclerosis, cirrhosis of the liver, and fibrosis of the kidney are often met with in the middle-aged who have succumbed to an attack. Healthy adults whose tissues are not spoiled by alcoholism, by laborious manual work, by overfeeding, or by disease seldom die. Obesity materially diminishes the chance of recovery.

3. **Degree of toxæmia and reaction of the patient to it.**—More than any other this factor determines the issue in a case of pneumonia. It is, of course, influenced by those already referred to—the virulence of the infection, the type of organism, the age and general condition of the patient—and by alcoholism.

The degree of toxæmia and the reaction of the patient can be estimated in various ways.

(a) **Fever.**—A moderately high fever is desirable, but if the temperature is persistently very high or shows marked irregularities the prognosis is less good. Much worse is a low fever, or an absence of rise of temperature altogether. Irregularity in the fever is of less moment in children than in adults.

(b) **The degree of leucocytosis** is an index of the severity of the infection and a measure of the resisting powers of the patient. Most favourable is a moderate increase of white cells—between 20,000 and 30,000. With a leucocyte count above 30,000 the mortality is greater, for it indicates a severe infection though one which is meeting with considerable resistance, and very high counts are not necessarily of evil omen. More serious is a feeble leucocytosis or a leucopenia; in such cases the patient is overwhelmed by his toxæmia, and the outlook is very grave. It must be remembered that the leucocytosis in children is generally greater than that in adults.

(c) **The condition of the circulation** is of great

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value in prognosis. A quick, feeble, irregular pulse, a weak cardiac beat, and weakness or absence of the pulmonary second sound are unfavourable. When the pulse-rate exceeds 130 the mortality is more than doubled. Evidence of failing circulation may be seen in an ashy-grey or dusky pallor. Fall of the blood-pressure as expressed in millimetres of mercury to below the pulse-rate expressed in beats per minute is a serious sign. This does not apply to children, in whom the blood-pressure is normally low, nor to the elderly or those with interstitial nephritis or arterio-sclerosis, in whom a high blood-pressure is ordinarily found. When these features are accompanied by coldness and blueness of the extremities, by clammy sweats and other signs of collapse, the immediate outlook is very black.

Engorgement of the right side of the heart, as indicated by increase of cardiac dullness to the right, distension of neck veins, enlargement of the liver, and marked cyanosis, is of less serious import than general cardiac and vasomotor failure, but materially adds to the gravity of the case.

(d) *Mental symptoms*.—An active delirium denotes a severe toxæmia, but is less ominous than the low muttering delirium accompanying the typhoid state, which is met with in the toxic or asthenic form of the disease. Delirium tremens is associated with a mortality-rate of about 80 per cent.

(e) The *respiratory rate* is largely controlled by the toxæmia, though other factors, such as anoxæmia and right-heart failure, clearly influence it. According to tables compiled by Prebles, the mortality is 50 per cent. or more when the respirations are above 50 per minute. A higher rate is to be expected in children than in adults.

4. **Extent of involvement of the lung**.—The amount of consolidation is of less importance than the degree of toxæmia, for a patient with only a small area of lung affected may succumb from the virulence of the infection, or may fail to resist one which is only moderate in severity. *Ceteris paribus*, affection of both lungs is more serious than that of one only, and affection of one lobe less serious than that of two or more.

5. **Certain complications**, other than those already discussed, interfere greatly with the chances of recovery. Most important in this respect is generalization of the infection, in which a fatal issue is common. When either malignant endocarditis or meningitis results,

death is to be expected. Pericarditis is another severe complication, though it is less fatal than endocarditis. Empyema delays recovery, but, when due to pneumococci, seldom determines a fatal result. If secondary infection by streptococci or other organisms is added, this complication is more troublesome; return to health is longer delayed, and the chances of ultimate recovery are considerably diminished. Pulmonary abscess and gangrene are unfavourable and generally fatal, whilst well-marked congestion or œdema of the lung which has escaped consolidation are also serious. In the early stages gastric dilatation may rapidly lead to collapse. Tympanites, by interfering with diaphragmatic movements, by hampering the heart, and by causing loss of sleep, militates against success in treatment. Diarrhœa, especially in children, is ominous.

Prophylaxis.—The discovery that pneumococci of Types I and II are generally absent from normal mouths, but are found with considerable frequency in the mouths of those who have been in contact with patients suffering from these infections, points to the spread of pneumonia by carriers. These organisms together are responsible for about 60 per cent. of all cases (Stillman). Auto-infection is the exception rather than the rule as was formerly supposed. It is important, therefore, to regard a patient with pneumonia as a possible source of further infection, and to isolate him to at least the same extent as in typhoid fever. The sputum should be received into a spittoon containing antiseptic, and all utensils should be kept apart and sterilized. Antiseptic measures should be employed to render the secretions of the patient's nose and throat innocuous, and after convalescence antiseptic mouth-washes and gargles should be continued to avoid self-reinfection or spread to susceptible persons. One who has had pneumonia should thereafter take more than ordinary care against chill.

F. S. Lister has obtained excellent results by using protective vaccines in the native mine labourers in the Transvaal. He found that a large proportion of the infection was due to one of three types of pneumococci, and subcutaneous inoculation of 1 c.c. of vaccine containing representative members of these types and having a total content of 7,000 million cocci per c.cm. rendered a large native mine population absolutely resistant to infection by organisms of any of these groups for a period of nine months. Definite responses have been

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obtained by vaccination to Types I and II of the American observers. It was most constant and greatest when 13,000 million cocci were injected. Owing to the toxic reaction, it is desirable to divide the dose between two or three inoculations.

Treatment.—The accepted treatment of lobar pneumonia is conservative and stimulant. Failure is predestined in some cases, whilst others get well after treatment of a most irrational kind. The issue of the conflict between the patient and his infection is, however, in many cases in the hands of his medical attendant and nurses, and in no disease is the physician more repaid for reasoned and assiduous care. Success depends upon conserving the patient's strength, by rest, by suitable dieting, by relieving symptoms and securing sleep, and upon stimulating when this is called for, anticipating the advent of circulatory failure rather than following in its train. It must be remembered that, in those cases in which treatment is most potent for good, the period of greatest stress and the height of the battle is in the hours preceding the crisis. Some bolts should be kept unshot for this period lest the patient fail to reach the crisis or die exhausted while it is in progress.

General treatment.—The patient's bed should be in an airy room with an equable temperature, and should be sheltered from draughts by a screen if necessary. Fresh air is essential, and the windows should be kept open throughout the twenty-four hours. Treatment in the open air is very successful if carried out with reason, but if combined with exposure of the patient and disregard of climatic conditions it is distinctly harmful. Chill must be avoided by warm clothing, blankets, and hot-water bottles when the atmospheric temperature is low. In the colder seasons a vest made of gamgee tissue, and tied at the side so that access to the chest is easy, is a useful garment in addition to the usual nightclothes. The position in bed should be that which gives the patient greatest rest, and will usually be found to be a semi-recumbent one. Generally, he will prefer to lie for most of the time on the affected side, but will be relieved by occasional changes in posture. In the case of children, particular care will be needed to avoid leaving the patient too long in one position.

A well-pillowed support for the head and shoulders and some means of preventing the patient from slipping down in bed are valuable adjuncts.

Dietetic.—Though the powers of assimilation are limited, the diet need not be restricted to milk. The addition of eggs beaten up in the milk, and of cocoa, custard, or broths, will help to keep up the strength. Beef tea, jellies, or Valentine's meat-juice are pleasant changes which may be permitted if sufficient food is being taken. Water, lemonade, or barley-water may be allowed plentifully to allay thirst and promote elimination. A common error in the case of children is to give only milk whenever a drink is called for, though there is usually ample opportunity of administering the desirable amount of milk as well as of allaying the thirst.

Local treatment.—Poulticing the chest is out of fashion, but there can be little doubt that properly made linseed poultices bring comfort and relief from pain. They should be light and changed frequently. A heavy poultice, or one which is allowed to become cold and clammy, does more harm than good. Poulticing should only be used for the relief of symptoms, such as pain and cough, which interfere with rest, for the necessary manipulations dissipate the patient's strength. If the ease which accrues fails to compensate for this, no benefit can be expected. Other forms of applying heat for a like purpose are by fomentations and by turpentine stupes. Either of these is more suitable for young children than poultices, for they are easier of application and lighter. In the case of turpentine stupes great care must be taken to avoid blistering; the skin should previously be anointed with vaselin or lanolin, and a watch kept on the local effect produced. It is not unusual to see patients whose chests have been badly scarred by mustard plasters or turpentine stupes ignorantly or carelessly applied during early life.

Local treatment by cold in the form of an ice-bag often relieves pain and may prove efficacious when heat has failed. The late Dr. D. B. Lees was firmly of opinion that ice-bags, properly applied, one in front and the other over the back of the consolidated area, have an inhibitory effect on the inflammatory process, hindering its development and preventing its extension. They should be placed directly on the skin. There is no doubt that they keep the fever within bounds, lessen the mental symptoms, and are often comforting to the patient. If they fail to bring relief they should not be persisted in. When ice is used, chilling of the patient must be prevented by blankets, and by hot-water bottles in the bed.

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Other measures of relief which are efficacious are cupping and leeches. The application of one or more leeches to the painful area sometimes produces immediate cessation of the pain.

Medicinal treatment consists in measures for the conservation of the patient's strength and the relief of symptoms. Expectorants are not generally called for, and the most useful mixture in the early stages is a simple saline, such as the following :

R̄ Pot. cit. ʒss.
Sod. nitr. gr. ss.
Liq. ammon. acet. ʒii.
Aq. camph. ad ʒi.
Quartis horis.

It acts as a mild febrifuge and eliminative.

The bowels should be opened by calomel 3 gr., followed next morning by a saline purgative mixture or by a seidlitz powder. Thereafter saline alone is usually sufficient.

The best guides to the need for *stimulation* are the character of the pulse and the blood-pressure. When the pulse increases in rate or becomes irregular or feeble, and when the blood-pressure steadily falls, stimulants are indicated. The most valuable are strychnine and digitalis. They should forestall failure rather than follow it. Strychnine hypodermically or by the mouth should be given, in all but mild cases, early in the disease, and may be pushed to as much as 10 min. of the liquor, hypodermically, if the condition of the circulation demands it. More than any other drug it counteracts the prostration and the exhaustion of nerve-centres which are to be feared. No drug more successfully steadies and slows the heart than digitalis; its action cannot be expected to be so potent as on healthy cardiac muscle, but it is nevertheless beneficial. It may be added in the form of the tincture (10-15 min.) to the strychnine mixture, or combined with strychnine hypodermically as digitalin ($\frac{1}{2}$ gr.). Caffeine is a valuable adjuvant, and also assists diuresis. The following prescription, recommended by Hale White, represents the degree of stimulation necessary in a severe case :

R̄ Tr. digit. ℥xv.
Liq. strych. ℥x.
Caffein. gr. v.
Sod. salicyl. gr. iiss.
Aq. ad ʒi.
Quartis horis.

Concerning the value of alcohol there is much difference of opinion. As a routine measure it is inadvisable, for the secondary depressant

effects probably counterbalance the initial stimulation. As the crisis approaches, if increased stimulation is needed, it is of great use and will help materially in tiding over the critical period. When its administration is decided upon it should be given freely, with due regard to the previous habits of the patient in this respect. Less than 5-10 oz. of whisky or brandy is useless in a patient who is addicted to spirits. If it increases the restlessness or produces insomnia it must be withheld.

Oxygen inhalations, if properly applied, are of undoubted benefit, but their effect should be carefully watched. Some patients experience rapid relief, their colour improving and breathing becoming easier. Others appear to be embarrassed by the treatment, and its application seems to increase their distress and meets with resistance; in these circumstances it should not be persisted in. It may be administered by a funnel, or more economically and potently by a mask. The conducting rubber tube should always be immersed in hot water so that the gas is warmed. The benefit is no doubt directly proportionate to the degree of anoxæmia. It has been found experimentally that an atmosphere containing about 50 to 60 per cent. of oxygen is the most effective.

Alcohol and oxygen treatment may be combined by passing the oxygen through absolute alcohol in an ordinary wash bottle. The alcohol vapour thus combined is non-irritating, and rapidly induces the therapeutic action of the drug, with no tendency to disturb digestion.

Symptomatic treatment. *Sleeplessness.*—To the patient, sleep is of first-rate importance, and failure to produce sleep for him is one of the gravest mistakes which can be made. The cause of the insomnia should be searched for and treated. If it be due to pain in the chest, local treatment with an ice-bag, fomentations, or leeches may be sufficient. Congestion of the right side of the heart, hyperpyrexia, and meteorism should receive appropriate treatment (*see below*). Tepid sponging, limb by limb, is a valuable means of inducing sleep. Such drugs as bromides, chloral, and paraldehyde may suffice, but opium is more to be relied upon. Regarded formerly as dangerous because of its depressant effect on respiration and circulation, it has won its way back into favour, for any depression is far more than counterbalanced by the sleep obtained. It may and should be used without hesitation if the usual remedies fail to induce sleep; in the early stages Dover's powders may be given, but later

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morphia hypodermically ($\frac{1}{4}$ gr.) is more certain. It may be combined with atropine ($\frac{1}{16}$ gr.) or with scopolamine hydrobromide ($\frac{1}{24}$ gr.).

Pain.—The treatment of pain has already been referred to. Local measures should be reinforced with morphia when necessary.

Pyrexia.—Antipyretic drugs are not advisable, for a moderate fever is less harmful. When the temperature soars above 103.6° F., tepid sponging is the best remedy, and generally causes a drop of a few degrees, together with a sense of comfort, and often induces sleep.

Dilatation and congestion of the right side of the heart.—In plethoric individuals, when cyanosis is marked and the right ventricle and systemic veins are engorged, venesection affords prompt relief. It is not necessary to remove a large quantity of blood, 5 oz. being generally sufficient. In children leeches are preferable. Three to six may be placed over the lower ribs on the right side, and fomentations applied subsequently to increase the amount of blood removed. The advantage of this situation is that the bleeding can readily be controlled by pressure if necessary.

Respiratory symptoms.—When the cough is painful and distressing, preventing the patient from resting, paregoric $\frac{1}{2}$ dr. may be given by the mouth, and, if necessary, morphine or heroin hypodermically, in addition to the usual local remedies. Edema of the lungs is best treated with strychnine and atropine.

Sudden cardiac failure and fall of blood-pressure indicates the use of rapidly acting stimulants, such as strychnine, strophanthin ($\frac{1}{16}$ gr.), camphor (3 gr. in 10 min. of olive oil), or caffeine-sodio-benzoate (5 gr.) hypodermically. Most valuable in this connexion is pituitrin, 1 c.c. of which may be injected deeply into the muscles or subcutaneously. Its advantages over adrenalin (1 : 1,000, 10 min.), which is also employed, is that the pressor effect is more abiding.

Tympanites may be ameliorated by turpentine stupes, by hot-water enemata, and by pituitrin.

Specific therapy.—Many drugs have from time to time been advocated as specific remedies. Among these are camphor, mercuric succinimide, and colloidal silver, all of comparatively recent date. Seibert recommends the subcutaneous injection of 10 c.c. of a 30-per-cent. solution of camphor in sesame oil for each 100 gm. of body-weight, every 8–12 hours. Wright claims to have aborted pneumonia by hypodermic injection of mercuric succinimide,

and has used $\frac{1}{4}$ gr. as a single dose. Netter uses colloidal silver prepared chemically or by electrical methods. He advocates inunction by an ointment containing 15 per cent. of colloidal silver, and injection of electargol or weak solutions of collargol, 0.25 gm. to the litre, subcutaneously.

Quinine urea hydrochloride 15 gr. has been injected intramuscularly every three hours and is said to have been beneficial in toxæmic cases (E. Matthew). Much was hoped from Optochin (ethyl-hydro-cuprein), introduced by Morgenroth, but its claims have not been substantiated.

Vaccine-therapy has some adherents. It is more likely to be useful when prepared from the patient's own organisms, but this necessitates some delay. Observation of a series of cases so treated has failed to convince the writer of its value.

Serums.—Recent work promises more success from homologous serums, but at present no satisfactory results have followed their use, except in pneumonia due to pneumococci of Type I. In 65 of these cases treated by the homologous serum and reported by Dochez, the mortality was 7.5 per cent., as compared with 25 per cent. in cases due to pneumococci of the same type and treated by ordinary means. Serum disease is, however, a frequent complication. For this treatment to become of practical use, a rapid method of determining the type of infection is essential.

II. BRONCHO-PNEUMONIA (LOBULAR PNEUMONIA, CATARRHAL PNEUMONIA)

An inflammation of the finer bronchi and of areas of pulmonary tissue around them.

1. PRIMARY BRONCHO-PNEUMONIA

This form occurs in children, especially in those under 2 years of age, during which period it almost displaces lobar pneumonia. So little is it related to the other forms of broncho-pneumonia that it ranks as a separate disease and will be considered apart. It is more nearly allied to lobar pneumonia, and is almost certainly due to infection by the pneumococcus alone or in preponderance. It occurs suddenly in children in good general health, perhaps after a chill. The temperature rises quickly and ends by crisis or by rapid lysis; it persists for five to nine days in most cases, and during its course varies no more than is commonly seen in cases of lobar pneumonia at this age.

The **symptoms** are those of lobar pneumonia

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in children. The onset is often marked by nervous phenomena such as head retraction, rigidity of the neck and of the limbs, photophobia, headache, delirium, and vomiting, and sometimes by convulsions. Meningitis may be so closely imitated as to make the diagnosis doubtful at first. Restlessness and thirst are manifest. The respirations increase out of proportion to the pulse, often exceed 60 and sometimes become as frequent as 100 per minute. The breathing is often inverted, the pause being ended by expiration accompanied by a grunt, which is followed by a rapid and short inspiration. The rhythm in these circumstances is, therefore, expiration—inspiration—pause, instead of the normal inspiration—expiration—pause. Dilatation of the *alæ nasi* more often accompanies expiration than inspiration. Considerable dyspnoea may be brought about by the action of the toxins upon the bulbar centre, but obstructive breathing is little in evidence, and cyanosis is generally slight. Though the respirations are rapid, there are not the distressing and ineffectual efforts to secure aeration of the lungs seen in broncho-pneumonia of the secondary forms. The heart may fail from toxæmia either directly affecting it or affecting the cardio-vascular centre, and become rapid, weak, and irregular, but there is seldom serious distension of its right side.

On examination of the chest a fairly large area of consolidation is found, apparently involving the whole of one or more lobes. In addition, there are smaller areas on the opposite side, and perhaps also in the remainder of the lung on the same side. The consolidation is a patchy one, but in one area, and sometimes in more than one, the patches are so aggregated as to simulate the uniform and continuous consolidation of lobar pneumonia.

Over the larger areas where they come to the surface the characteristic signs of consolidation are heard, but when the areas are small many of the signs may be lacking; thus there may be dullness, harsh breathing, and crepitations only, or impaired note, diminished breath-sounds, and increased vocal resonance, or harsh breathing and increased vocal resonance, or, again, only sharp crepitations. Over deeply-seated areas of consolidation there may be a total absence of signs. The degree of bronchitis is slight, and little or no secretion is coughed into the throat. The disease is a self-limited one, and with the termination of the fever the child's condition changes from

distress to comfort, and the signs in the lungs clear up. Though there may be delay or failure of resolution, and recrudescences are not rare, true relapses are exceptional. They may, however, occur again and again, and be separated by intervals of as long as a week or ten days.

The **prognosis** is more favourable than in secondary broncho-pneumonia, but the disease is more often fatal than is the lobar form met with in children over 2. Many of the deaths are due to generalization of the infection, which is commoner than in pneumococcal pneumonia after infancy.

Diagnosis.—The chief difference from lobar pneumonia clinically is the patchy bilateral distribution. Post mortem it is seen that the consolidation is grouped around bronchi and that the large area of consolidation which simulates lobar pneumonia is merely an aggregate of peribronchial patches. The cut surface seldom has the granular appearance of the lobar form, and histologically it is found that the alveolar exudate is more largely composed of cells, catarrhal and leucocytic, and less of fibrin.

The **treatment** is that suitable for lobar pneumonia (*see above*).

Briefly, it may be said that the symptoms of this form of pneumonia are mainly those of toxæmia due to the pneumococcus, and hence resemble those of lobar pneumonia, that the physical signs are different only because the patches are multiple, that the diagnosis, prognosis, and treatment are those of lobar pneumonia except in so far as they are influenced by the age, but that the histology is that of broncho-pneumonia, with the reservation that the degree of bronchitis is slight. The consolidation is not postbronchitic, as in the secondary forms.

2. SECONDARY BRONCHO-PNEUMONIA

Etiology.—Secondary, consecutive, or post-bronchitic pneumonia occurs under the following conditions:—

(1) As a complication of infective diseases, especially those accompanied by catarrh of the respiratory passages. Thus it is a common complication of measles and whooping-cough, which two diseases provide the greatest number of examples of this group. It is also frequently met with in influenza, diphtheria, and scarlet fever, and less often in smallpox, typhus and typhoid fevers, erysipelas, and other infections.

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(2) In the form of aspiration and deglutition pneumonias due to inhalation of septic material or irritating gases from the upper air-passages, or spread of septic infection along the bronchial tree. Included in this group are most of the septic broncho-pneumonias.

Here must be mentioned the broncho-pneumonia which follows septic conditions of the mouth and pharynx, and operations in this area. The source may be dental sepsis, quinsy, retropharyngeal abscess, suppuration of the accessory air sinuses, or new growth of the tonsils or pharynx, whilst a common surgical antecedent is an operation for carcinoma of the tongue. The primary lesion may be situated in the larynx, as laryngeal ulceration, simple, syphilitic, tuberculous, or malignant. Laryngotomy and tracheotomy, suicidally or surgically performed, are other causes. Bronchial sources are suppurative bronchitis and bronchiectasis. Carcinoma of the œsophagus by invading the air-passages, and aneurysm or mediastinal glands or neoplasms by obstructing them, are occasional causes. An empyema which ruptures into a bronchus is a rare precursor. Foreign bodies in the air-passages, if not removed, usually cause death by septic broncho-pneumonia, for they introduce septic micro-organisms and, by obstruction, prevent the expectoration of the purulent exudate which follows.

When the larynx is paralysed or insensitive, as in postdiphtheritic paralysis, bulbar palsy and other forms of vagal paralysis, and in coma, food and liquid are prone to reach the air-passages and, by causing broncho-pneumonia, determine a fatal issue. In the same way broncho-pneumonia is a cause of death in patients who have been rescued from drowning. Aspiration broncho-pneumonia may follow in the train of hæmoptysis. In ordinary times the irritating gas which is most in evidence in this connexion is ether, for the larger proportion of postanaesthetic pneumonias is lobular in type. The poison gases of war have provided many examples of inhalation broncho-pneumonia.

Other etiological considerations. *Age.*—Broncho-pneumonia is prone to occur at the extremes of life. The age-incidence of measles and whooping-cough makes it very usual under 5 years of age, 75 per cent. of all cases of broncho-pneumonia in children falling within that period. In the aged it is a common harbinger of death, following bronchitis or influenza, or terminating some chronic or

acute disease in individuals already enfeebled by years.

Season.—The late autumn, winter, and spring months are those in which the greatest number of cases occur. This applies especially to the broncho-pneumonia which complicates measles, whooping-cough, and other specific infections.

Hygiene.—Broncho-pneumonia is prone to attack the children of the poor; overcrowding, ill-ventilated rooms and insufficient clothing co-operate in its genesis. The habit of leaving the legs and arms of small children bare to inclemencies of the weather plays a part. Not unimportant is the undue coddling to which those children are subjected who are confined for the greater part of their time in hot, stuffy rooms with shut windows, and go abroad only on warm days. On the other hand, failure to realize the gravity of measles or of whooping-cough and imperfect care of the child during the course of these infections or during convalescence account for many cases.

Previous health.—Marasmic infants and those prostrated by epidemic diarrhoea are very liable to attacks. Rickets not only predisposes to the disease but jeopardizes the issue. The weakly and enfeebled at both extremes of life fall easy victims. In the elderly, chronic debilitating diseases such as nephritis, cirrhosis of the liver, and diabetes render their owners vulnerable. Hypostatic congestion often results in a true inflammatory lesion; the latter is therefore liable to occur in chronic valvular or myocardial disease and in patients with feeble circulation who are confined to bed. Such diverse disorders as fracture of the femur in the elderly and paralytic nervous diseases thus play an etiological part in broncho-pneumonia.

Tuberculous broncho-pneumonia is considered separately (*see* PULMONARY TUBERCULOSIS).

Bacteriology.—Whilst primary broncho-pneumonia is probably, at least in its inception, a pure pneumococcal infection and blood-borne, the secondary form is due to a variety of infections and is consecutive to bronchitis. Pneumococci are found in about half of the cases in children, but usually in association with other organisms. Streptococci and staphylococci are probably the chief invaders if all ages be taken into consideration, for they are the dominant organisms in the septic forms. Staphylococci are seldom present alone, but streptococci often constitute the only recognizable infection of the lung, especially in measles and whooping-cough. Other organ-

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isms found are *Micrococcus catarrhalis*, *Micrococcus tetragenes*, the bacillus of Friedländer, *Bacillus influenzae*, *Bacillus diphtheriae*, *Bacillus typhosus*, *Bacillus pertussis* and *Bacillus pyocyaneus*. In an investigation of 95 cases Eyre found that of this latter group only the bacillus of Friedländer and that of influenza could be obtained in pure culture.

Pathology.—Many components go to make up the post-mortem appearance of the lung in broncho-pneumonia. Chief among these are areas of *consolidation*, which stand out from the surface, are firm to the touch, friable, and sink in water. Though scattered irregularly and almost always bilateral, they are found more frequently and to a greater extent towards the pulmonary bases. On the surface of larger areas the pleura is injected, lustreless and roughened, and may be covered with an inflammatory exudate of fibrin or pyo-lymph. Surrounding the solid areas, or sometimes placed discretely, are patches of lung tissue, somewhat less firm to the touch, less prominent, and red in colour; these represent consolidation at an earlier stage, and have been spoken of as areas of splenization or carnification.

Section shows that these patches of solid and semi-solid lung are grouped according to the ramification of the bronchi. The cut surface of the mature patches is greyish or yellowish grey in colour and usually smooth, though granulations produced by distended alveoli may be met with. The cut surface of the more immature patches is redder and drips blood.

Three types of consolidation are recognized as occurring in different cases. The commonest is that in which areas of varying size and maturity are scattered irregularly through the lung. In a second, the pseudolobar form, the patches are so aggregated that little or no normal lung intervenes and the greater part of a lobe is consolidated. The consolidation is not uniform, however, for strands of unaffected lung separate the solid areas into groups, and varying stages of maturity are encountered. The third is distinguished by an apparent absence of consolidation at first sight. Section reveals minute areas around the bronchi, perhaps uniformly distributed throughout the lungs. Into this group fall the cases which were formerly termed "capillary bronchitis"; "miliary broncho-pneumonia" more correctly describes them. In severe and rapidly fatal examples the consolidation is only microscopic in degree.

In the non-solid parts of the lung patches of the *collapse* and of *emphysema* are met with. The

areas of collapse are bluish or dark red in colour and depressed. The bases of the lobes are most commonly affected in this way, while the *emphysema* is often most marked at their apices and anterior margins. The collapsed portions may be inflated through a blow-pipe. Areas of either may be seen in close proximity to those which are solid. The collapse is due to blocking of the corresponding bronchus by mucus and to the defective aeration of the whole bronchial tree, while the throwing out of function of large portions of the lung and the forced character of the inspirations sufficiently explain the distension of the alveoli and production of *emphysema*.

Bronchitis is an important feature. The mucous membrane of the bronchi is swollen and injected and lustreless from loss of endothelium, and the lumina are filled with mucus or pus. On section this exudate can readily be squeezed out of the bronchi to form little yellow pools on the cut surface of the lung. Sometimes the bronchioles become dilated—*bronchiolectasis*. Later, perhaps, as the result of coughing, the softened walls of the dilated bronchi may rupture, the pus within them extravasating into the surrounding pulmonary tissue. In this way the lung becomes filled with cavities and an appearance to which the apt name of "honeycomb" lung has been given is produced. Larger *abscesses* and *gangrene* are also met with, particularly in aspiration and deglutition pneumonias.

Microscopically, each constituent part of the lung is seen to be involved, bronchi, alveoli, and interstitial tissue being affected. The inflammation is revealed as spreading from the bronchioles, each individual patch of consolidation having a bronchiole for its centre, about which the inflammatory changes are at their most advanced stage. The bronchus is dilated and filled with pus or mucus, containing shed and swollen epithelial cells, and there is cellular infiltration and swelling of its walls. The alveoli immediately around the bronchus are filled with leucocytes and epithelial cells. Red corpuscles are often seen, and may be abundant. A fibrin network may be detected, though it is not so conspicuous as in lobar pneumonia, while the shed alveolar (catarrhal) cells are in greater abundance. The alveolar walls are swollen and infiltrated with round cells, and their capillaries are dilated and engorged. The farther the bronchus is left behind, the less intense is the inflammation.

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Symptomatology. — The *onset*, though rapid, is not abrupt as in lobar pneumonia. The patient is generally under observation for one of the numerous precursors of the disease, or perhaps is convalescent from measles or whooping-cough. There has been some cough and shortness of breath and signs of bronchitis have been detected, perhaps accompanied by some fever. In other cases there is merely a history of a "severe cold" followed by cough and dyspnoea. The first evidences that the infection has spread to the lung are a rapid rise of temperature, accompanied by increase in pulse-rate, greater dyspnoea, and a more urgent and distressing cough. The patient is distinctly ill. On examination of the chest at this stage signs of bronchitis are heard, with patches of weak breath-sounds and sharp fine râles. With the progress of the disease the dyspnoea becomes more severe, the pulse more rapid, and the lips cyanosed. The cough is distressing, sometimes paroxysmal, and pus or mucus is brought up, to be swallowed in the case of children, or expectorated in adults. Restlessness increases. Signs of patchy consolidation may now be detected in the lungs, and moist sounds, fine and coarse, are heard. After a few days or a week or so the temperature may fall by lysis, the symptoms rapidly improve coincidentally with disappearance of the signs, and convalescence begin. On the other hand, the respirations may fail and the cough become ineffectual or cease, while cyanosis increases and the fever persists. Mucous râles are more abundant and generalized. The pulse fails, pallor and sweating supervene, and the patient sinks into coma and dies. In other cases, after a short illness the fever ceases and the symptoms and signs improve, but after a brief apyrexial interval a new rise of temperature heralds a fresh patch of consolidation. In some relapse follows relapse until the course of the disease is protracted into several weeks, perhaps to end in recovery when the outlook appears hopeless.

Fever.—The onset is marked by a rise of temperature. Thereafter the pyrexia is very variable, ranging in an average case from 102° to 105° F. Fluctuations may occur daily or at more irregular intervals. After persisting for a week or ten days, sometimes for a shorter period, the fever in a favourable case gradually falls by lysis. The temperature chart is of considerable importance, for it follows fairly closely the condition of the lung, dropping slowly as a patch of consolidation resolves and

rising again with the development of new pneumonic areas. Relapses or recrudescences are thus depicted on the chart. With recrudescence the fever rises to a higher level without having attained the normal, perhaps without having shown any disposition to fall; in relapses there may be an apyrexial interlude of twenty-four hours or longer before the next rise of temperature. In fatal cases a terminal hyperpyrexia is not unusual. In feeble individuals, and especially in weakly marasmic infants, there may be no fever throughout a fatal illness. Defervescence by crisis is probably an indication of a pure pneumococcal infection, and is more often seen in primary broncho-pneumonia.

The *pulse-rate* is increased, and depends upon the pyrexia, the degree of pulmonary obstruction, the condition of the heart and the severity of the toxæmia. It often attains 120 per minute, and may exceed 180 or become uncountable. The pulse is frequently irregular both in rate and in volume, especially in cases in which asphyxia is most in evidence.

Dyspnoea is an obvious symptom. It is more obstructive in type than in lobar pneumonia, and is less related to the toxæmia. The respirations soon increase to 40 or 60 per minute and may exceed 100. To aerate its lungs the child fights for its breath, sitting up in its endeavour if old enough. The accessory muscles come into play and are prominent. In infants the upper part of the chest often protrudes, and recession of the lower thoracic border and of the intercostal spaces may be seen with each inspiration. Dilatation of the *alæ nasi* accompanies inspiration. The pulse-respiration ratio may be modified, becoming 3:1 or 2:1. Grunting expiration may occur and the respiratory rhythm may be inverted, expiration preceding inspiration, though this is less common than in lobar pneumonia. The obstructive nature of the breathing is well exemplified by the results of a bout of coughing or vomiting; these partially empty the air-passages, and a decrease in the urgency of the dyspnoea and improvement in colour is noticeable. Toxæmia of the bulbar centres sometimes leads to irregular or grouped respirations.

Cyanosis results from the obstruction of the air-passages, the consolidation and collapse, and the imperfect pulmonary circulation. Like the dyspnoea, it is sometimes extreme, the lips and periphery becoming purplish or mauve-coloured and sometimes almost black.

Coughing is a more troublesome and per-

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sistent feature of broncho-pneumonia than of lobar pneumonia. In the early stages it is dry and frequent, sometimes painful. Later it occurs in longer bouts, is moist, and often paroxysmal. With failing respiration and increasing cyanosis the cough becomes weaker and ineffectual, and soon ceases altogether.

Expectoration is scanty at first, but soon becomes more abundant, and consists of frothy muco-pus or pus. It is less tenacious than in lobar pneumonia and seldom rusty, though considerable quantities of bright-red blood are met with in some cases. Infants and young children seldom expectorate, but the use of a spatula will often provoke an attack of coughing and the appearance of the characteristic sputum in the back of the throat.

Other symptoms are want of appetite, thirst, and restlessness. At first the child cries when a bout of coughing begins, but with diminishing strength and greater dyspnoea this is replaced by moaning. The acute cerebral symptoms which have suggested the name "cerebral pneumonia" are seldom seen in secondary broncho-pneumonia, but with failing oxygenation and increasing cyanosis, drowsiness, coma, and perhaps convulsions supervene and anticipate death. Retching and vomiting often accompany the cough.

The skin is usually hot and dry, but sweating is sometimes marked; towards the end the skin becomes cold and clammy and covered with beads of sweat.

Physical signs.—In the early stages little beyond the signs of bronchitis can be detected on examining the lungs. Râles, coarse and medium, can be heard, while the breath-sounds are harsh, though still vesicular. Soon the characteristic fine crepitations indicating involvement of the finer bronchioles make their appearance. They are generally heard towards the bases of the lungs, but other patches, irregularly distributed and usually bilateral, are found also. They vary considerably from time to time, new patches appearing whilst others diminish or disappear. These crepitations with harsh breath-sounds may be the only signs of consolidation met with throughout the illness. In military broncho-pneumonia (capillary bronchitis) they may be heard over the greater part or the whole of both lungs, death ensuing before any patch of consolidation large enough to provide its usual signs has been produced.

In the majority of cases some of the patches of crepitations and harsh breathing, sooner or

later, manifest more or less completely the ordinary signs of consolidation, dullness being associated with bronchial or tubular breathing and increase of vocal or cry resonance and fremitus.

In some areas amphoric breathing with loud crepitations, bronchophony and a "cracked-pot" note may be obtained, suggestive of excavation. They are most pronounced when there is considerable bronchiolitis and peribronchial suppuration. It must be remembered, however, that such signs do not necessarily indicate a cavity in an infant or a young child.

The dullness in broncho-pneumonia is seldom so complete as in lobar pneumonia. A hyper-resonant note due to emphysema may be obtained over unaffected portions of lung, especially at the apices. Recession of intercostal spaces with impaired percussion note and feeble breath-sounds may indicate collapse, and are most commonly found at the extreme bases. In some cases the bronchitic signs are so loud as to mask all other auscultatory signs, though perhaps these may again be elicited after an attack of coughing.

In the *pseudo-lobar variety* of broncho-pneumonia the aggregated patches provide an area of dullness, considerable in extent, and in this area the clinical signs are then more defined and similar to those of the lobar form of the disease. There are, however, patches elsewhere on the same side or in the other lung, broncho-pneumonic in form. This pseudo-lobar variety is less often met with in secondary broncho-pneumonia than in the primary form.

There may thus be found in a case of broncho-pneumonia many different and varying groups of signs over the lungs—(1) signs of bronchitis of larger and smaller tubes; (2) signs of consolidation at various depths and at various stages: these include fine crepitations with harsh breath-sounds, frank signs of consolidation, signs of excavation and signs of resolution; (3) signs of pulmonary collapse, and (4) signs of emphysema.

Complications.—*Pulmonary bronchiolectasis* with multiple suppurative foci in the lung has been mentioned. A considerable *abscess* or *gangrene* is rare except in the aspiration or deglutition pneumonias. Want of resolution and subsequent *fibrosis* of the lung sometimes occur: it is especially common in the broncho-pneumonia complicating whooping-cough, and a considerable proportion of the examples of *fibrosis* and *bronchiectasis* met

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with in later years date from this disease in early life.

Pleurisy occurs over larger superficial patches of consolidation, but is not a prominent feature, and is seldom detected during life. Effusion, whether serous or purulent, is much less common than in lobar pneumonia.

Cardio-vascular complications.—The strain upon the right heart imposed by the embarrassed respirations and by the hindrance to pulmonary circulation often leads to right-heart dilatation. The dullness increases to the right, and is accompanied by hepatic enlargement, visible engorgement of the neck veins and increase of the cyanosis. The effect of toxæmia on the heart-muscle is seen by feebleness and rapidity of the pulse, and in severe cases by attacks of pallor, during which the pulse is almost imperceptible or temporarily lost. Recovery may ensue in a case in which these phenomena occur, but they are indications of a very grave condition.

Nervous complications.—In some cases, especially in infants, the respirations become cyclical, periods of apnoea, in which the colour becomes worse and the pulse slower, being interposed. The respiratory rhythm may be Cheyne-Stokes in kind, or the respirations may merely be grouped. During an apnoeic phase the pulse may not be felt, and this sign, together with lividity, cessation of the respirations and stupor, seems to portend the end. Breathing then starts again and improvement follows. Such cases may terminate favourably, though the outlook seems devoid of hope. These attacks, and possibly also those of sudden pallor, appear to be bulbar in origin.

Drowsiness deepening to coma, and convulsions, are generally late events in a fatal case.

In adults active delirium with a tendency to get out of bed, and delirium of a low muttering type, occur in severe cases. Suicidal tendencies are not very unusual. During the epidemic of influenza in 1917-19 the writer saw three cases of cut-throat in as many days in delirious broncho-pneumonic patients. Delirium tremens is an occasional complication. Meningitis is not common.

Alimentary-tract complications.—Diarrhoea is a serious complication in infants, and often the immediate cause of death. Vomiting is also commoner in children; it is of less moment and often decidedly beneficial by helping to clear the air-passages of muco-pus. Flatulent distension of the stomach and intestines may

hamper breathing considerably. Infection of the naso-pharynx with profuse purulent discharge from the nose is often seen in children; there is considerable danger of extension of the infection along the Eustachian tubes and the production of otitis media. Suppurative or gangrenous parotitis is an occasional complication in adults.

Diagnosis.—From *simple bronchitis* there is usually little difficulty. Broncho-pneumonia is accompanied by a higher temperature and greater toxæmia. The patient is more ill than in bronchitis and the dyspnoea is generally more urgent. The sharp crepitations audible over the lung are characteristic, even though no area of dullness or bronchial breathing is detected. Much of the difficulty has arisen in connexion with so-called "capillary bronchitis," which is really a variety of broncho-pneumonia. In old and feeble subjects and in marasmic infants absence of fever may cause the pneumonia to be overlooked.

The chief differences between secondary broncho-pneumonia and *lobar pneumonia* may be given in tabular form (*see* p. 583).

Lobar pneumonia is sudden in onset, defined in course and abrupt in termination, while secondary broncho-pneumonia is more gradual in onset, has an indefinite and often protracted course and ends gradually. The symptoms of the former are mainly those of toxæmia; those of the latter are largely due to mechanical interference with aeration. The physical signs in lobar pneumonia are those of one or more large areas of consolidation, lobar in distribution; in broncho-pneumonia the consolidation is patchy and bilateral and its signs are irregular and often ill-marked and accompanied by evidences of bronchitis.

In old patients and in others with failing circulation it may be impossible to distinguish between the two diseases; the signs of lobar pneumonia may be ill-defined and the reaction to toxæmia almost lacking, while oedema of the lung provides râles or rhonchi in abundance.

The diagnosis between *tuberculous* and *non-tuberculous* broncho-pneumonia is attended with great difficulties and may be impossible, especially in children. In adults the aspiration and deglutition varieties are not likely to be confused with tuberculosis; in children it may not be possible to say whether a patchy consolidation following measles or whooping-cough is tuberculous until the autopsy. Discovery of tubercle bacilli in the sputum settles the diagnosis, but sputum is not always

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DIAGNOSIS BETWEEN LOBAR PNEUMONIA AND SECONDARY BRONCHO-PNEUMONIA

Lobar Pneumonia

Rare under 2 years.
Sudden in onset.
Generally a primary disease.

Often attacks patients in robust health.

Temperature rises abruptly, is maintained, and generally ends by crisis.

Breathing shallow and painful and largely abdominal, dyspnoea less urgent and orthopnoea rare.

Signs of consolidation lobar in distribution.

Bronchitis absent clinically.
Sputum tenacious and rusty.

Duration 5-10 days, and relapses rare.

Complications: empyema and pneumococcal infection elsewhere prone to occur.

Secondary Broncho-pneumonia

Common under 2 years.
More gradual onset.
Proceeded by bronchitis and generally secondary to other diseases.

Prone to attack the feeble and weakly at both extremes of life.

Temperature rises gradually, is irregular, and falls by lysis.

Dyspnoea obstructive in type and laboured; accessory respiratory muscles in action; orthopnoea common.

Signs of consolidation patchy, bilateral, and often ill marked. The patches in different stages of development.

Bronchitis well marked.

Sputum more abundant, and purulent or muco-purulent.

Duration indefinite; often protracted and marked by relapses.

Empyema and generalization of infection rare.

obtainable. Aggregation of the consolidation at the apices and a regularly remittent temperature favour but do not denote tuberculosis. In the tuberculous form there is generally more rapid loss of flesh. A family history of tuberculosis, or the presence of tuberculosis in the house, may help in the differentiation. Replacement of signs of consolidation by signs of softening, with excavation in the dull areas, points to tuberculosis.

The form of tuberculosis most nearly resembling secondary broncho-pneumonia is acute general tuberculosis, in which the lungs are studded with miliary tubercles. The signs in the lungs are practically identical. As a rule, the toxæmia is more pronounced in this form of tuberculosis and prostration occurs early, before the occurrence of obstructed breathing, whereas in broncho-pneumonia the prostration follows a period of increased respiratory efforts. The detection of tuberculosis elsewhere, as, for example, choroidal tubercles, may establish the diagnosis.

Prognosis.—Broncho-pneumonia is one of the most serious infective diseases, and is fatal in about half the cases; if one excludes primary broncho-pneumonia the mortality is probably considerably higher. The aspiration and deglutition pneumonias are especially dangerous, and in them a fatal termination is usual.

Age is an important factor. Under one year the mortality-rate is about 80 per cent.; at the other extreme of life the disease is generally fatal.

Previous health.—Marasmic, feeble infants and those with epidemic diarrhoea, and adults debilitated by Bright's disease, cancer, tuberculosis, diabetes, etc., usually succumb. Broncho-pneumonia affecting a patient weakened by influenza or typhoid fever or following an operation is particularly dangerous. In a child, rickets renders the outlook more serious since it seriously hampers the respiratory efforts. The broncho-pneumonia which complicates diphtheria is very ominous, and that complicating whooping-cough but little less so.

Toxæmia. When the toxæmia is profound, so that prostration and stupor occur almost from the onset of the disease, death is to be expected. In such cases the pyrexia is often slight, and may be lacking. The same is true when the patient is too feeble to react to the disease.

Frequent relapses indicate a good resistance to the infection, and such cases may end in recovery even after a duration of some months. Each relapse, however, exhausts the child's strength, and the relapsing type is therefore necessarily of serious import.

Extent of lung involvement.—*Celeris paribus*,

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extensive consolidation of both lungs increases the gravity of the disease. One large area with little consolidation elsewhere is less serious than are several small areas in both lungs. Particularly fatal is the miliary form, in which great difficulty in breathing and severe toxæmia are accompanied by signs of bronchiolitis.

Symptoms and complications.—Cyanosis, stupor, great pallor, convulsions and diarrhoea are all serious symptoms, and render the outlook relatively grave. A rapid, feeble pulse, quickening as time goes on, is a warning of danger. Cessation of coughing, with accumulating moist sounds in the air-passages, presages a fatal issue. The combination of lividity, stupor, delirium, plucking at the bedclothes, cold sweats, and noisy breathing indicates that the end is approaching.

Prophylaxis.—The most effective prevention of the common form met with in children is proper care during convalescence after the specific fevers, especially measles and whooping-cough. Too little care is paid as a rule to these diseases, the child often being allowed up almost directly after the fever abates. The lungs should be examined carefully from day to day, and the patient not allowed up for at least a week after all bronchitic signs have disappeared. In weakly and marasmic infants the occurrence of catarrhal signs in the bronchi should always be regarded as a possible precursor of broncho-pneumonia, and the infant be confined to its cot in a warm room, and exposed as little as possible. Persons suffering from a "feverish cold" and from influenza should forthwith take to their beds, and remain there until at least a week after the temperature has dropped and the signs of catarrh have disappeared.

Attention to the mouth, nose, and throat during specific fevers will do much towards lessening the risk of broncho-pneumonia. Similarly, before surgical procedures, oral and naso-pharyngeal sepsis should be dealt with whenever possible. This is particularly important when tracheotomy or laryngotomy is a stage in the operation.

Broncho-pneumonia as a complication of the acute specific fevers of childhood, especially of measles and whooping-cough, should be regarded as a contagious disease demanding isolation. There is little doubt that when it arises under these conditions its virulence is greatly enhanced, and it is readily transmitted to other children in the same ward or room.

In this country, with its rapid changes of

climate, there is no question but that the habit, seen chiefly among the poor, of leaving the legs of young children almost bare is conducive to this disease.

Treatment.—The general treatment is similar to that of lobar pneumonia (p. 574). Rest in bed, a warm room, warm clothing and abundance of fresh air are indicated. Treatment in the open air is satisfactory if the weather is equable and the patient is kept sufficiently warm in bed, but no procedure which requires exposure such as physical examination should be undertaken except in a warm room. Treatment on a balcony or in the garden is particularly valuable in the obstinately relapsing cases, and often effectually stems the relapses. When treatment is undertaken indoors, which is usually the only method practicable, the windows should be kept open day and night, the bed being screened from draughts where necessary.

Much controversy centres round the *steam kettle and tent*, and they have largely gone out of use. Though generally unnecessary and often inadvisable, they are valuable under special conditions. When there is difficulty in breathing in the early stages, with dry catarrhal signs in the lungs and a frequent, painful and harassing cough causing loss of sleep, the relief they afford is incontestable. The steam kettle should not be employed for longer than about half an hour at a time, and care should be taken that the temperature of the room is not allowed to fall, lest the resulting cold damp air more than counteract the good effect of the steam. When the râles become moist and the air-passages are laden with mucus and pus, the steam kettle and tent should be abandoned.

During the early stages, too, when the bronchial mucous membrane is dry and expectoration absent, light poultices or turpentine stupes are of great value in relieving distress and in controlling the painful cough. Gentle rubbing of the chest with a liniment containing turpentine, I regard as distinctly beneficial; the sense of warmth gives comfort and sleep to the patient, and possibly also inhalation of the fumes is not without benefit. A suitable liniment consists of equal parts of linimentum terebinthinæ and olive oil. Warm packs and warm sponging are very valuable procedures, reducing restlessness and promoting sleep.

The diet is necessarily a fluid one, for the mastication and swallowing of solid food are

distressing to the patient, and gastric and intestinal disorders are serious complications which should be kept at arm's length. Milk will form the staple diet during the acute stage; it may be peptonized, citrated, or diluted with barley water. In young infants albumen water only, or veal or chicken tea, should be given if there is want of appetite or looseness of the motions. The demand for fluids should not be ignored, and water or barley water or, in the case of adults, lemonade may be given freely. When the appetite is good the milk may be thickened with a patent food or groats, or, in the case of older children or adults, eggs may be stirred in.

Medicinal. — Expectorants are useful, but they must be employed rationally in accordance with the bronchial signs. In the early stages, when the râles are few and dry and expectoration is difficult or absent and accompanied by a harassing cough, diluent saline expectorants, such as ammonium carbonate or chloride, ammonium acetate and potassium citrate, are most beneficial. Ipecacuanha wine may be added at this stage. Later, when abundance of moist râles make their appearance, stimulant expectorants, such as squills and senega, are indicated. Should the accumulation of muco-pus become excessive, so that breathing is increasingly asphyxial and cyanosis deepens, an emetic is called for, especially in the case of children, but should not be employed if the heart is feeble or collapse threatens. The old-time administration of a drachm each of ipecacuanha and antimony wine seldom produces emesis nowadays, possibly because of their greater purity; apomorphine hypodermically ($\frac{1}{16}$ gr.) is more certain. Belladonna is a useful drug when bronchial secretion is abundant, for it lessens the exudation, generally diminishes the coughing, and stimulates the respiratory centre. It may be employed in the form of the tincture (3–10 min. every four hours according to age), or hypodermically as atropine (5–10 min. of the injection for adults). In infants it is advisable to use the injection diluted to 1 in 4. When belladonna is employed its therapeutic effect should be watched carefully, and if it increases the patient's discomfort by drying up the mouth and bronchial secretion, administration should be stopped. Paregoric or heroin often allays the troublesome ineffectual cough; these drugs must be employed with caution, and are contraindicated if the bronchial tubes are filling with muco-pus.

The state of the circulation requires very careful watching. There is no drug so valuable for warding off or combating failure as strychnine. Its employment should not be deferred too long. Within the first few days it may be given by the mouth as the tincture of nux vomica. When signs of impending or existing failure are present it should be given hypodermically as the injection (5–10 min. for an adult or 1–4 min. for a child) every four or six hours, and may be pushed until reflexes become exaggerated. Digitalin or strophanthin may be combined with the hypodermic injection of strychnine.

Alcohol, in my opinion, is of considerable value, whether its action be that of a direct cardiac stimulant or whether it acts chiefly by sparing the tissues or cutting off peripheral stimuli. It is especially indicated in the presence of restlessness and loss of sleep, and is also particularly useful in feeble infants and old people, whose vitality is low and whose powers of digestion and absorption are slight. For children 10–30 minims of brandy according to age, and for adults half an ounce of whisky, may be given every two or three hours. (While these spirits remain at war-time strength, double this quantity is required.)

When engorgement of the right side of the heart becomes manifest, depletion is called for. It is best attained in children by the application of three to six leeches over the right lower ribs, reinforced by fomentations if necessary, and in adults by venesection. Excellent results in such cases follow the removal of quite small quantities of blood (5 ounces in adults); improvement in colour and in breathing, and sleep are thus obtained.

Anoxæmia plays so important a rôle in broncho-pneumonia that the use of oxygen inhalations is based on a sound foundation. The combination of alcohol and oxygen, procured by bubbling oxygen through absolute alcohol contained in a wash bottle as recommended by Willcox, is of undoubted value. It may be administered continuously for ten minutes at the first application and thereafter for five minutes every half-hour or hour as indicated.

The highly infective condition of the mouth and naso-pharynx should not be ignored, but should be attacked by antiseptic lotions, such as listerine, borol (1 : 4), or carbolic acid (1 : 40). It is a possible source of reinfection and a frequent forerunner of persistent purulent rhinorrhœa or otitis media.

The gradual invasion of new areas of lung

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and the frequent occurrence of relapses in a patient who is gradually losing strength call for great pertinacity and attention to detail on the part of the medical attendant. In no disease are his acumen and resource more tried; but it can be said also that in no disease are his ministrations more amply rewarded.

In **convalescence** the patient must be safeguarded from chill and exposure; too early a return to ordinary life is a common danger. Reviving appetite demands a liberal dietary. Iron and general tonics are indicated. In the case of children the iron may usefully be combined with cod-liver oil or malt. Parrish's food or collosol ferromalt is valuable at this stage. A sojourn for three months in a sunny climate or at the seaside should be procured whenever possible. This is especially important in the case of children, in whom incomplete resolution and tuberculosis of the mediastinal glands or lungs are very liable to supervene.

FREDERICK LANGMEAD.

PNEUMONIC PHTHISIS (*see* PULMONARY TUBERCULOSIS).

PNEUMONOCOONIOSES. -- The various forms of inflammation, acute or chronic, which affect the lungs as the result of dust inhalation are known as the pneumonocoonioses (a term of Greek origin, *πνεύμων*, lung, and *κόνη*, dust). The subject may be dealt with by grouping known facts either according to various kinds of dust, or according to the diseases which result. The latter method, which, from the medical point of view, is the more scientific, is followed here. The diseases of the lungs which have been recognized to follow upon inhalation of different dusts are, asthma, bronchitis, pneumonia, and dust-phthisis.

Asthma.—This term, which indicates a train of symptoms rather than a pathological disease entity, has been somewhat loosely used in relation to dust troubles. In this connexion it should be confined to the occurrence of spasmodic asthmatic attacks associated with (i) exposure to a peccant dust and ceasing on leaving the dusty atmosphere; (ii) immobility of the diaphragm; and (iii) over-action of the superior intercostal and extraordinary muscles of respiration. It should not be applied to the shortness of breath on exertion exhibited by the subjects of advanced pulmonary fibrosis; this latter form of dyspnoea is not set up by exposure to dust, during an attack the action

of the diaphragm is noticeable, while the other respiratory muscles are immobile. True dust-asthma has been best described among flax-hecklers and cotton-strippers; these workers used to be exposed to the inhalation of dust composed of vegetable husk (flax and cotton), and the condition, though less marked, may occur among other operatives exposed to dusts of similar origin, such as wood bark, jute, and the harder woods. Dust-asthma takes from five to twenty years, according to the idiosyncrasy of the worker, to develop. An individual affected then presents the well-known appearance of a chronic asthmatic, with rounded shoulders, emaciated frame, prominent eyes, and laborious wheezing respiration. At first the operative is only troubled on starting work on Monday, and gets better as the week progresses; but as years go by the trouble extends later and later into the week, until he is compelled to seek other employment.

Asthma seldom appears on death certificates, but in the past cotton-strippers have suffered a higher mortality from pneumonia and bronchitis than other cotton operatives. Differential diagnosis of asthma due to dust is only to be made through the history of the case and knowledge of the exact industrial exposure of the patient. The presence of the peccant dust in the sputum may be of assistance.

Carefully devised methods of dust removal have almost abolished this trouble from the textile industries. Its occurrence in the past has, however, indicated its causation and origin, so that mild cases in these and other industries can be recognized and steps taken for their prevention.

True dust-asthma has not been described as associated with the inhalation of any other form of dust than those mentioned above.

Dust-bronchitis.—Bronchitis as caused by dust stands *par excellence* first among the pneumonocoonioses. Dust which causes any other form of respiratory disease also causes bronchitis. Its importance, however, is liable to be overlooked because (i) dust-bronchitis is indistinguishable clinically from bronchitis due to other causes; and (ii) it is a complaint which advances slowly and, though causing incapacity, does not cause much mortality during the working period of life. When comparative morbidity statistics for different occupations are available, this disease will stand out as the chief cause of invalidity in dusty occupations. Certain forms of dust undoubtedly, by over-stimulation of the bronchial mucosa, sensitize the air-passages.

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to microbial invasion. Among these dusts are quartz, flint, dust of sandstone, vegetable husk of cotton, flax, jute, and hemp, certain hard woods, emery, glass, slag-wool, and basic slag. The common properties of these materials are (i) such brittleness as leads them to break into fine dust; (ii) insolubility in the normal secretions of the air-passages; and (iii) non-plastic, i.e. non-colloidal structure. Dusts which have not been found associated with bronchitis are pure coal dust, plaster-of-paris, alabaster, limestone, pure clay, cement, and dusts of animal origin such as bone, leather, and horn.

The rôle played by dust in irritating the walls of the larger air-passages is more conspicuous than the part played by subsequent microbial infection, although both contribute their quota in establishing a condition of chronic bronchitis. When present in an individual in whom dust-inhalation has caused pulmonary fibrosis (see below), or the barrel-shaped chest typical of asthma, the physical signs of these conditions may indicate the connexion between dust-inhalation and the disease. Otherwise a diagnosis of pure dust-bronchitis cannot be made from study of any particular patient, but knowledge of exposure to dust and of the known effect of this dust in causing bronchitis will assist, especially if the dust in question is found in the sputum.

The degeneration of the bronchial mucosa and the condition of emphysema which occur coincide with those described for ordinary bronchitis elsewhere in this work and require no repetition here. After death, dust particles will not often be detected; for the disease is slow in its nature, causing prolonged incapacity, and recent exposure to dust-inhalation must, therefore, be exceptional.

Dust-pneumonia.—The influence of dust-inhalation in causing pneumonia is not sufficiently appreciated. Even those who hold that this disease is a general fever with local pulmonary manifestations, must allow that the local manifestations indicate the *locus minoris resistentie* especially affected by the disease. Certain dusts, particularly that of basic slag, have been recognized to be associated with a high prevalence of pneumonia among those who inhale them. Such dusts in order to reach the alveoli of the lungs (the site of pneumonic manifestations), must be small enough to pass through the finer bronchioles, i.e. below 6μ in diameter; dusts larger than this only gain access to and affect the larger

air-passages, when bronchitis results. When allowance for this question of size of particles is made, all those dusts—and no others—which are associated with the occurrence of dust-bronchitis are found also to be associated with a high prevalence of pneumonia. But in this disease the rôle played by dust is less conspicuous than that of the microbial infection for which it paves the way.

The influence of dust, as described so far, may be summarized thus: Any influence which interferes with the vitality of the air-passages increases the probability of microbial invasion, whether that influence be alcohol impairing ciliary action, or cold, or traumatism, either acute, due to local injury, or chronic, due to dust-inhalation. In fact, the part played by dust is to sensitize the air-passages to invasion.

One dust has a peculiar influence in relation to pneumonia, viz. silica dust. Inhalation of this dust is in early years associated with a high prevalence of bronchitis and pneumonia; in later years the prevalence of bronchitis persists, but that of pneumonia is entirely replaced by phthisis. The reason most probably is that silicotic fibrosis obliterates to a great extent the alveoli and bronchioles which are the areas concerned in pneumococcal inflammation.

Dust-phthisis (see also INDUSTRIAL MEDICINE).—This disease is the most distinctive of the pneumonocoineses; its study has led to recognition of the part played by various dusts in causing other respiratory diseases. The history of this condition, which is only known to occur among persons exposed to the inhalation of silica dust, probably dates back to the dawn of human industry. Human industries started in prehistoric factories for the manufacture of flint implements, and the lineal occupational representatives of this industry, the flint-knappers of Brandon, have been shown to suffer to-day a terrible mortality from this disease, amounting to over 30 per 1,000 per annum.

In other industries where this disease is prevalent, as for instance among grinders of metals or sandstone masons, the mortality ascribed to phthisis is often as high as 15 per 1,000 living per annum, as compared with 2.5 for other males; while 50 per cent. of all deaths instead of 12 per cent. are ascribed to phthisis (the grouping under which deaths from dust-phthisis appear).

Silica occurs in nature as flint, quartz (in

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sandstone and granite), quartzite (in gneiss and buhrstone), rock-crystal, and quartzose sand.

The path followed by particles of dust of any kind small enough to gain access to the pulmonary alveoli has been closely traced; some, probably the majority, encapsulated in cells are returned to the bronchioles to be expelled in sputum, but others are drawn in through the pseudo-stomata into the lymph channels, when they are either deposited in the trabeculae of the alveoli, carried to the pleural surface, or removed to the lymph-glands. There is no evidence (unless massive experimental amounts are inhaled) that the presence of dust particles in these positions interferes with physiological functions: if the particles can be absorbed by the body fluids they quickly disappear; if they cannot be absorbed they are more slowly eliminated, either by being returned to the alveoli or by removal elsewhere; the capacity of the lungs for getting rid of dusts is considerable. Only in the particular case of silica dust, if sufficient is inhaled over sufficiently long periods, does a distinctive reaction occur. First, due to the peculiar chemical properties of silica, the parenchyma of the lungs is converted into fibrous tissue, when a pathological condition known as pulmonary silicosis results; this stage in industry takes, on an average, nine years to develop. Secondly, tuberculous invasion of the damaged tissues takes place, when the condition becomes one of tuberculous silicosis. The stage of silicosis before infection occurs is distinctive; it depends upon the gradual conversion of portions of the lungs into fibrous tissue with complete obliteration of the alveoli and small air-passages. The change is usually bilateral; it commences at the apices, spreading downwards, and starts first in the pleural coverings, which become thickened and the site of firm pleuritic adhesions. The patient after a time begins to find himself out of breath on exertion, and his chest expansion is found to be greatly diminished. Diaphragmatic breathing is distinctive of the condition. Uncomplicated silicosis may advance until the heart is overtaxed in carrying on the pulmonary circulation, when oedema of the legs and ascites usher in a fatal termination from heart failure; such cases are rare. Infection with tubercle usually intervenes and rapidly carries off the patient. The picture of pulmonary tuberculosis which results only differs from that more usually seen in so far as the fibrotic state of the lungs invaded is peculiar; hæmoptysis is a rather unusual complication.

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Here, as in the case of pneumonia and bronchitis, dust acts as a sensitizer to microbic invasion, but the action is a chemical one and is due to the formation in the parenchyma of the lungs of silicotic fibrous tissue which tubercle bacilli easily attack. One unexplained characteristic of this form of tuberculosis is that it shows an unusually low power of infecting contacts.

Note should be made that dangerous silica dust, when inhaled with fireclay dust, does not appear to be followed by tuberculous silicosis. The reason for this inhibitory action of fireclay has not yet been ascertained. It should be noted also that the dust of silicates does not lead to the formation of silicosis.

This disease has a legal importance, since on the Rand gold mines in South Africa it is the subject of a special compensation scheme, and in this country it is being made a reason for compensation as the provisions of the Workmen's Compensation (Silicosis) Act, 1918, are applied to the different industries concerned.

Conclusions.—1. All dusts are not equally dangerous to inhale.

2. Dusts are more injurious as their chemical composition differs from that of the human body, or from the elements of which the body is normally composed.

3. Dusts which are dangerous exert their influence by sensitizing different parts of the respiratory system to microbic invasion.

4. No dust of animal origin is known to be harmful.

5. Dusts of husks of vegetation cause trouble, which takes the form of asthma and is associated with a high mortality from pneumonia and bronchitis.

6. Inorganic dusts if soluble in the fluids of the body are not harmful.

7. Insoluble inorganic dusts set up inflammatory conditions of the air-passages, giving rise to pneumonia and bronchitis.

8. One form of inorganic dust only, viz. silica, not only gives rise to pneumonia and bronchitis but also causes the formation in the lungs of a form of fibrosis, known as silicosis, which is particularly sensitive to attacks of tubercle bacilli.

EDGAR L. COLLIS.

PNEUMOTHORAX.—The presence of air or other gas in the pleural cavity.

Etiology.—Pulmonary tuberculosis probably accounts for nearly 90 per cent. of the cases. It results especially from acute phthisis, owing to the softening of a caseous mass situated

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close to the pleura before protective adhesions have formed. Sometimes it occurs at a very early stage in patients who have had no suspicion of pre-existing disease. Such cases are comparable with those in which hæmoptysis is the first recognizable symptom of phthisis. In some instances it is apparently due to rupture of an air vesicle from muscular strain, but it is difficult, if not impossible, to exclude a small tuberculous lesion, unless, as may possibly happen, an emphysematous bulla has ruptured. Even this must be very rare, as these bullæ, fragile though they look, are well supported by the chest-wall. A number of other diseases of the lung occasionally give rise to pneumothorax, e.g. a gangrenous or septic infarct, an abscess or hydatid cyst, and bronchiectasis; also the bursting of an empyema into the lung (see EMPYEMA). Malignant disease of the lung or of the œsophagus may produce it. It may also arise from external causes, as the result of a perforating wound of the chest or of a fractured rib wounding the pleura and lung. In such cases, however, a pneumothorax does not usually follow, because the force of cohesion between the two layers of the pleura is greater than the elastic traction of the lung tending to separate them; subcutaneous emphysema is a more likely consequence.

Pathology.—The opening from the lung into the pleural cavity may be the size of a pin's point only, or large enough to admit the tip of the little finger. It is generally closed in a few days by inflammatory lymph. It may be quite free, or valvular; in the latter case, air enters the pleural cavity with each inspiration, but is prevented from escaping during expiration; it therefore accumulates under great pressure and causes correspondingly severe symptoms. When the pulmonary lesion giving rise to a pneumothorax is a very slight one, as in early tuberculous disease, in those cases in which it has apparently arisen from muscular strain, and when it is due to fractured rib, the air which enters the pleural cavity is filtered in its passage through the lung and rendered practically aseptic. In such cases, as soon as the opening in the lung has closed (and this may occur in a day or two), absorption of the contained air follows and in a few days the lung re-expands completely. When, however, as a result of the causal lesion many septic germs enter the pleural cavity, as, for instance, from the rupture of an old phthisical vomica, acute inflammation is set

up and an effusion follows, which may be either sero-fibrinous (hydropneumothorax) or purulent (pyopneumothorax). Should the germs be of a very virulent type, as in cases due to septic infarct, gangrene of the lung, or cancer of the œsophagus, the pus may be excessively fetid. Many cases of pneumothorax, especially those due to phthisis, are more or less localized by pre-existing pleural adhesions.

Symptomatology.—The onset is usually sudden, with a feeling of something having given way, and severe pain in the side, especially if inflammation is produced. There is dyspnoea, which, in the case of a valvular opening and accumulation of air under pressure, may become intense, with cyanosis, collapse, and fall of temperature. Later on, should pus form, an irregular pyrexia will probably develop. It is important to realize, however, that the symptoms may be comparatively slight, perhaps quite unobtrusive, should the pneumothorax occur when the patient is already seriously ill, as in an advanced stage of phthisis; or if the condition is localized.

Physical signs.—The affected side is usually distended, and moves but little—if at all—with respiration; the heart is displaced to the healthy side; vocal fremitus is diminished or lost. If the air in the pleural cavity is under pressure the intercostal spaces may bulge and the liver or spleen be pushed down, according to the side involved. On percussion, the note is more or less tympanitic, but this may be less marked in cases of extreme distension. If effusion, which may appear in the course of a few hours, be present, there is dullness, which moves freely with every change of posture of the patient, like that of ascites, thus differing greatly from the dullness of an ordinary pleural effusion. A considerable quantity of fluid, as much even as two or three pints, may, however, be present with but little dullness in cases in which the diaphragm is pushed down, for when the patient sits up the fluid lies in a cup-shaped depression formed by that muscle, and but little of it is in contact with the chest-wall. Again, if the patient be examined in the recumbent position, it follows, from the free movement of the fluid, that when he is on his back there is resonance in front, and when he turns over on to his face the back becomes resonant; consequently it is easy to miss the effusion altogether, unless it is fairly large. On auscultation, breath-sounds are usually absent if the

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pleural cavity is full of gas ; but if the opening in the lung is free they may be extremely amphoric. Distant amphoric breathing transmitted through the pneumothorax may also be heard if the lung is not collapsed completely but expands a little with inspiration. In such cases the voice and cough-sounds may also be amphoric or metallic in character. Lastly, three special signs are often though by no means invariably present : (a) The " bell or coin sound " produced by percussing a coin apposed to the affected side of the chest by another coin, while the observer listens over another part of the pleural cavity ; a characteristic, clear, ringing sound or metallic echo (*bruit d'airain*) may be heard. (b) The " succussion splash," a sound produced by fluid splashing in the pleural cavity when the patient is gently shaken ; hence obtainable only when an effusion is present, and more readily with clear fluid than with pus. (c) The " metallic tinkle," a peculiar echoing sound like that produced by a drop of water falling in a cave or grotto, due probably to the bursting of bubbles in a large space filled with air.

Diagnosis.—This may be exceedingly easy ; when, for instance, a patient is taken suddenly ill with severe dyspnoea and a sharp pain in the side, and on examination the heart is found much displaced, one side of the chest fixed and over-distended, with a hyper-resonant percussion note and no breath-sounds, the diagnosis is perfectly obvious. Yet the very fact that diagnosis is sometimes such a simple matter renders mistakes all the more easy to make in less straightforward cases. In a large proportion of patients the history, the symptoms, and the physical signs are by no means characteristic. When the pneumothorax is localized, it is obvious that difficulties are likely to arise ; also when the condition develops in one who is suffering from advanced phthisis it may easily pass unnoticed, the patient slowly sinking without showing any marked or abrupt change, and being so ill that no careful physical examination is either desirable or possible.

Even when a complete pneumothorax occurs in an individual previously in good health, it is remarkable how comparatively slight and transient may be the symptoms if the other lung be perfectly healthy ; so much so that the possibility of pneumothorax may hardly be entertained. Another difficulty in diagnosis, perhaps the most important of all, is the extraordinary variability in the physical signs. The

affected side, instead of being over-distended, may even be somewhat contracted when there is a perfectly free opening from the lung into the pleural cavity ; and the respiratory movements, instead of being diminished or lost, may be quite free, air passing directly in and out of the pleural cavity without distending the lung. When the patient is first seen at a comparatively late stage, dullness rather than hyper-resonance may be the most marked feature on percussion, and the presence of a large effusion may be diagnosed quite correctly while the pneumothorax is overlooked. In some cases, as already mentioned, breath-sounds and voice-sounds are altogether absent, whilst in others there is amphoric breathing and bronchophony. Lastly, the three special auscultatory signs previously described, though very significant, rarely occur together, and, although one of the three can usually be detected, no single one is absolutely pathognomonic. The coin sound may be heard over a large cavity, the succussion splash may be obtained shortly after a patient with a dilated stomach has drunk some water, and the metallic tinkle is a very inconstant phenomenon. Recognizing these difficulties, it is well to note those features which are most reliable in arriving at a diagnosis. Displacement of the heart is particularly important, but is sometimes very difficult to make sure of, for the impulse may be feeble or imperceptible and the cardiac dullness masked by over-distension of the healthy lung. A hyper-resonant percussion note is always present to a greater or less extent, and its limitation to one side serves at once to distinguish it from the hyper-resonance of emphysema. Shifting dullness is most significant of the presence of air and fluid together in the pleural cavity. The breath-sounds, however much they may vary in character, are always abnormal and obviously different from those heard over the healthy lung. Sometimes radiography enables an immediate diagnosis to be made, especially when fluid is present, and by it the size of the effusion can be determined with greater accuracy than by any other method.

Sometimes it is difficult to distinguish between a large cavity in the lung and a localized pneumothorax, but with the former there is not likely to be any displacement of organs, and the note is hardly ever definitely hyper-resonant. A traumatic rupture of the diaphragm, with displacement of the stomach into the pleural cavity, may give rise to physical signs

indistinguishable from those of a pneumothorax, the more so as the state of the patient may not admit of a very satisfactory examination, and the accident may well be responsible for either condition. In doubtful cases, unless the patient is profoundly collapsed, it is best to open the abdomen, for he can hardly be made worse by an exploration, and there is at least the possibility that it may save his life.

When the presence of a pneumothorax has been recognized, its cause has still to be considered. Speaking generally, it may be said that unless some other fairly obvious cause is present the condition should be regarded as due to tuberculous disease of the lung.

Prognosis.—This naturally varies with the cause. So-called simple cases, apparently due to muscular strain, and occurring in patients previously in good health, do well; for, as already explained, no inflammation is set up and the air is completely absorbed in a few weeks or even days. Such patients, however, must always remain suspect of tuberculosis; they are in much the same position as a man who has suffered from an unexplained pleurisy, or from an attack of hæmoptysis for which no cause can be discovered.

When pneumothorax forms in an advanced case of phthisis it is a not infrequent cause of death, occasionally in a few hours or even minutes, if the other lung is much diseased and the pneumothorax develops rapidly and completely, more often in a few days or weeks, the patient never really rallying, but going steadily down hill. Even if he survives, a condition of hydro- or pyo-pneumothorax often persists. On the other hand, there is always the possibility that the development of a pneumothorax may check or arrest the progress of the disease in the compressed lung. When the condition is due to some septic disease the outlook is obviously very grave.

Treatment.—In most cases the initial pain and shock first call for treatment; hot fomentations to the side and stimulants may be required, but a hypodermic injection of morphia is often the most effective measure. Should dyspnoea be great and persistent, and especially if there are indications of a valved opening with excessive intrapleural tension, the air should be let out by aspiration or simply by the introduction of a small trocar and cannula; the side afterwards may be fixed by strapping. In some cases it is desirable to leave the cannula *in situ* for a time, covering it with an antiseptic dressing.

If effusion occurs, a little of the fluid should be drawn off with an exploring needle, to determine its nature. If it is clear it may be removed by siphonage or aspiration; but if after two or three withdrawals it continues to re-accumulate, as is often the case, it is well to leave it, or only to remove it should the quantity become very large. The same plan may be adopted even if it be purulent, so long as there are no indications of constitutional disturbance; in such cases life may be prolonged for some years. Should, however, definite symptoms arise, or should the pus be fetid, the only course is to treat the patient as for an empyema and open and drain freely. Unfortunately, the lung is not likely to re-expand, a large pus-producing cavity remains, and the ultimate prognosis is grave.

J. WALTER CARR.

PNEUMOTHORAX, ARTIFICIAL (*see* PULMONARY TUBERCULOSIS).

POISONOUS FOODS (*see* POISONS AND POISONING).

POISONS AND POISONING.—A poison is a substance which by its direct action on the mucous membrane, tissues, or skin, or after absorption into the circulatory system, can injuriously affect health or destroy life. This definition includes such substances as powdered glass, metallic filings, etc., which would act mechanically as irritants if swallowed; it also includes substances which are of a harmless nature but which, by reason of the excessive quantity in which they are administered, or of the form in which they are given, may act injuriously on the body—for example, water at the boiling temperature.

The administration of any poison *with intent* to murder or cause grievous bodily harm, or to produce a stupefying effect whereby some indictable offence may be committed, or to cause abortion, is regarded by the law as felony.

Criminal poisoning—i.e. the administration of a poison with intent to murder or cause grievous bodily harm—is punishable by a very heavy sentence, and by death should a fatal result ensue.

The administration of a poison with the intent to aggrieve, injure, or annoy is a misdemeanour, so that practical joking with poisonous substances is punishable by law.

The sale of poisons.—The sale of poisons to the public is carefully controlled by law.

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With certain exceptions it is restricted to duly qualified and registered pharmacists and to medical practitioners.

The Pharmacy Act of 1868 divides poisons into two groups.

Part I includes the more powerful poisons, such as arsenic and its medicinal preparations, tartar emetic, all poisonous vegetable alkaloids, prussic acid, etc. For the sale of the substances in this group registration of the sale in a poison register before purchase is necessary, and the purchaser must be known to or introduced by some person known to the seller.

Recently veronal, medinal, and similar substances have been added under the title of "diethyl barbituric acid and derivatives of barbituric acid, and all poisonous urethanes and ureides." Other recent additions are lead plaster (diachylon), and preparations or admixtures containing 0.2 per cent. or more of morphine, or 0.1 per cent. or more of cocaine (ecgonine), or 0.1 per cent. or more of diamorphine (heroin).

Part II, with its recent additions, includes a large number of poisons such as carbolic acid or preparations containing more than 3 per cent. of it or its homologues; chloral hydrate, chloroform, sulphonal, sulphuric, nitric, and hydrochloric acids, oxalic acid and its soluble salts, and preparations containing more than 5 per cent. by weight of ammonia; also carbolic disinfectant preparations containing less than 3 per cent. of phenols, etc.

The sale of poisons in Part II need not be registered, but all poisons in both Part I and Part II must be labelled with the name of the article, the word "poison," and the name and address of the seller.

Special Regulations have been made by the Secretary of State for controlling the manufacture, sale, possession, and distribution of morphine, cocaine (ecgonine), and diamorphine (heroin) and their respective salts, medicinal opium, and preparations and substances containing not less than 0.2 per cent. of morphia or 0.1 per cent. of cocaine (ecgonine) or diamorphine.

The sale of poisonous substances such as arsenic or tobacco preparations used for agricultural or horticultural purposes is permitted by licensed persons who are not qualified pharmacists; regulations for the sale of these substances are carefully laid down, and a register of their sale has to be kept.

The sale of cocaine and opium has been carefully regulated by the Cocaine and Opium

Proclamation, 1916, in which all preparations of cocaine containing more than 0.1 per cent. are included. The sale of these substances to the public is prohibited except by a medical prescription, which must be dated and signed by the practitioner with his full name and address and marked "Not to be repeated"; the quantity of the medicine ordered is to be specified, the prescription is to be retained by the pharmacist, and a record of it made in a special book kept for the purpose.

Registered dentists and veterinary surgeons are permitted to order cocaine and opium under the above conditions.

An Army Council Order (1916) under the Defence of the Realm Act controls the sale of narcotic drugs to any member of H.M. Forces (except registered medical practitioners, dentists, or veterinary surgeons). The list includes veronal, chloral, cocaine, eucaïne, morphine, heroin, codeine, opium, cannabis indica, sulphonal, and all preparations, derivatives, or mixtures of these. The conditions of sale are similar to those under the Cocaine and Opium Proclamation.

Conditions affecting the action of a poison. 1. **The amount taken.**—Usually the quantity of a poison taken bears a relation to the effects produced; exceptions to this rule are substances in which, owing to the quantity taken, vomiting occurs so that most of the poison is expelled. Oxalic acid and tartar emetic are examples.

The term "fatal dose" when applied to a poison means the smallest amount which is known to have caused death in an adult. Frequently larger doses may be taken without death resulting, but much depends on other factors than quantity; another element is, of course, the treatment applied.

2. **Habit.**—A tolerance of some poisons occurs after their repeated use, and in some cases very large doses, greater than a normal fatal dose, may be taken without serious symptoms developing. Examples of such drugs are morphine, opium, cocaine, alcohol, and arsenic. In the case of some of these poisons their repeated use leads to the development of the "drug habit" with all its pernicious symptoms and effects.

3. **Idiosyncrasy.**—Some persons are exceedingly intolerant of certain drugs, which in moderate doses may cause in them dangerous or even fatal symptoms; examples are salicylates and aceto-salicylic acid, which in some subjects have a severe cardio-depressant action.

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Quinine may occasionally cause deafness and amaurosis in susceptible persons; I have seen almost complete temporary blindness follow a dose of 15 gr. Belladonna, atropine, and arsenic are further examples. Perhaps the most striking instance of this idiosyncrasy is the toxic effect upon certain persons of ordinarily harmless articles of food or of certain forms of pollen.

4. **Age.**—Usually children are much more susceptible to the effects of a poison than adults. The dosage of drugs in the case of children has been fixed by a pharmacological rule, but exceptions to this are morphine, opium and its preparations, which are much more toxic than the rule would indicate. On the other hand, children tolerate belladonna preparations better than adults. Aged persons withstand poisons badly.

5. **State of health.**—In disease drugs are usually much more toxic than in health, and this is especially so where the excretory organs are diseased; for example, in nephritis medicinal doses of such drugs as morphine, salvarsan, hyoscine, etc., often cause dangerous symptoms. In cirrhosis of the liver there is a greatly increased susceptibility to these drugs.

In some conditions associated with delirium or pain large doses of sedative drugs produce little effect, provided that the excretory organs are healthy.

6. **Mode of administration, etc.**—If a poison is taken by the mouth in solution it acts much more powerfully than if in an insoluble form. Thus, an insoluble preparation of arsenic may have little poisonous effect even in large doses; similarly, strychnine given in hard pills has a much delayed action.

The presence of food in the stomach has a marked influence on the effect of a poison. If the stomach is empty the effect will be rapid, particularly when the poison is in a soluble form; on the other hand, if the stomach is full, considerable delay may occur in the action of the poison, as is well shown in acute arsenical poisoning.

Poisons administered subcutaneously or intravenously act more powerfully than when given by the mouth.

When a poison is inhaled in the form of spray, vapour, or gas its absorption from the respiratory tract is rapid and the effect great. Poisons may be absorbed by the skin or mucous membrane of the vagina or rectum with fatal result.

Evidence of acute poisoning.—1. The

symptoms are usually sudden in onset. When the poison has been taken in food or drink, the interval varies with the kind of poison taken.

2. If several people take the same food mixed with poison, all are affected with similar symptoms. An exception to this rule occurs in bacterial food poisoning, the so-called ptomaine poisoning, in which only susceptible persons may be attacked.

3. The analysis which should always be carried out in suspected cases will reveal the presence of the poison in the food or the medicine, and in the vomit, and probably also in the urine and faeces.

Post-mortem evidence.—The post-mortem examination in cases of suspected poisoning should always be made in the most careful manner and every precaution be taken. A coroner's order is necessary, and, should the case be one of suspected homicidal poisoning, more than one medical practitioner should be present.

The examination should be made in good daylight with suitable facilities. No disinfectants should be allowed, since these are poisonous and may complicate the subsequent analysis if they come into contact with the body. No smoking should be permitted, since this will interfere with the detection of poisons by smell.

The examination should be a most thorough and careful one, every organ of the body, including the brain and spinal cord, being examined, so that any possible disease as the cause of death may be excluded.

Special attention should be paid to the examination of the alimentary tract, and when the stomach and intestines are opened this should be done in clean dishes and none of their contents should be lost. A careful note is made of the presence of any signs of irritant or corrosive poisoning in the gastro-intestinal tract, and also of signs of disease in any of the organs.

A number of clean jars should be ready for the reception of the various organs, and should be duly covered, labelled, and sealed, and taken charge of by the person responsible for their care, a receipt being obtained. The organs that should be reserved for analysis are—

The stomach and its contents.

The intestine and its contents.

The liver, spleen, and kidneys.

The heart and lungs.

A portion of the muscle.

The brain.

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Any urine found in the bladder should be reserved.

If arsenical or metallic poisoning is suspected, a piece of the femur should be reserved, and also portions of hair.

In exhumation cases a portion of the soil adherent to the coffin should be kept for analysis, and special note made of the conditions of burial.

No preservative of any kind should be added to any of the materials reserved for analysis.

Conduct of the medical practitioner in a case of suspected poisoning.—The utmost tact and discretion is required. An incorrect accusation would involve the medical practitioner in very serious difficulties, while, on the other hand, he is responsible for the life of the patient and must take due steps to safeguard it. He will be wise to ask for a consultation with another specially qualified practitioner in such a case, and also to obtain permission for examination of the vomit and excreta, though he would be wise in certain cases not to say what was the object of the analysis. He should insist upon nurses on whom he can rely being in attendance, and, when possible, the removal of the patient to a nursing home or hospital may be called for. The greatest care must, of course, be taken to make no accusation of poisoning unless there is unequivocal evidence.

Classification of poisons.—Poisons may be classified in many different ways, e.g. according to their chemical composition, their action on the body, their physical characters, etc. The following is a convenient and simple classification :—

1. **Corrosive poisons** are those that destroy by direct action the tissues with which they come in contact. They are the mineral acids, such as sulphuric, hydrochloric, and nitric acids; the caustic alkalis, such as caustic soda, caustic potash, and ammonia; carbolic acid; metallic poisons, such as corrosive sublimate, zinc chloride, and silver nitrate: these latter will be considered under irritant poisons.

It should be remembered that corrosive poisons in sufficient dilution lose their corrosive effect and become irritants.

2. **Irritant poisons**, by their direct action on the mucous membrane, set up inflammation. Examples are oxalic acid and its soluble salts, arsenic compounds, antimony compounds, most of the metallic poisons in solution, phosphorus, bromine, iodine, boric acid, etc.

3. **General poisons** act on the nervous sys-

tem or other important organs such as the heart, liver, lungs, or kidneys without having any special irritant or corrosive effect. This group comprises the majority of poisonous substances, such as the vegetable poisons or their alkaloids, hydrocyanic acid and its salts, chloral, chloroform, alcohol, ether, and hypnotic drugs (veronal, sulphonal, etc.).

Among the *liver poisons* are included those which cause toxic jaundice—tetrachlorethane, trinitrotoluene, picric acid, etc.; and among *renal poisons* are cantharides, turpentine, etc.

4. **Gaseous poisons** include chlorine, carbon monoxide, carbon dioxide, etc.

5. **Poisonous foods**, e.g. mushrooms, shell fish, and food contaminated with dangerous pathogenic bacteria

1. CORROSIVE POISONS

Symptoms.—Corrosive poisons produce severe symptoms immediately they are taken—a burning pain in the mouth, throat, and œsophagus, and pain referred from the stomach and intestines. Vomiting occurs quickly, and the vomit contains blood which may be altered in colour by the action of the poison; often also shreds of destroyed mucosa are present. Collapse occurs early, and perforation is common, which, should the patient survive, is followed by signs of general peritonitis.

An examination of the patient will reveal signs of the corrosive action of the poison in the mouth and throat, and great tenderness will be present on palpation over the stomach and intestines. Corrosive poisons, if swallowed in poisonous quantity, are usually fatal within twenty-four hours unless prompt treatment with a suitable antidote is adopted.

Should the patient survive the immediate effects of the poison, serious after-effects result owing to the damage done to the alimentary tract; in the case of volatile corrosives, serious pulmonary complications often ensue.

Post-mortem examination affords evidence of the destructive action of the poison on the mucous membrane of the mouth, throat, œsophagus, and stomach, and there is often extensive hæmorrhage in the underlying tissues.

CORROSIVE MINERAL ACIDS

Sulphuric acid (commonly known as oil of vitriol) is used in various industries and in all chemical laboratories. It is a most powerful corrosive in the concentrated form, and may be taken internally, or be used for criminal

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purposes externally in the form of "vitriol-throwing"—a not uncommon crime in France.

Fatal dose.—1 dr. has caused death in an adult, and half that quantity in a child.

Owing to its dehydrating effect, blackening often occurs in the tissues with which it comes into contact.

Hydrochloric acid (muriatic acid, spirit of salts) is used largely for industrial and chemical purposes and is a common article of domestic use. It is readily obtainable and is very commonly used for suicidal purposes.

It is a most dangerous corrosive, and 1 dr. has caused death in an adult.

Nitric acid (aqua fortis) is used for industrial and chemical purposes and is another very dangerous corrosive, producing characteristic yellow staining of the tissues with which the strong acid comes in contact (xanthoproteic reaction).

The vapour of the acid, if inhaled into the lungs, often produces an acute fatal form of pneumonia and thus provides a common cause of death in nitric-acid poisoning.

Fatal dose.—2 dr. have caused death in an adult.

Other mineral acids, such as hydrofluoric, phosphoric, and sulphurous acids, in concentrated form produce similar effects to the above.

The **treatment** of poisoning by corrosive mineral acids consists in giving freely, and as soon as possible, such harmless alkaline remedies as magnesium powder, lime-water, sodium bicarbonate, or chalk; these should be given freely. Plenty of egg-albumen (white of egg) should also be administered, since it tends to neutralize the acid by forming a protein combination, and also has a soothing effect on the damaged mucosa. The stomach should not be washed out, for fear of perforation. Pain is relieved by the free use of morphine hypodermically, and, after the swallowed acid has been neutralized, food should be withheld by the mouth and normal saline given per rectum as freely as it can be retained.

The *vomit* should be reserved for chemical tests.

The presence of free mineral acid is shown by the blue colour that is given with congo-red paper, or by the red coloration with dimethyl-amido-azo-benzene solution. The best test is Günzberg's (see GASTRIC CONTENTS, EXAMINATION OF).

The particular acid present is detected by the usual chemical tests.

CAUSTIC ALKALIS

Caustic potash (potassium hydrate or potash lye) is a powerful corrosive, and **potassium carbonate** (salt of tartar) has a similar though less powerful effect. Both of these are used industrially.

Fatal dose.—40 gr. of caustic potash have caused death.

Caustic soda (sodium hydrate or soda lye), also a powerful corrosive, is quite as dangerous as caustic potash. It is in common use industrially.

Ammonia (liquid ammonia or spirit of hartshorn) is used largely for domestic and industrial purposes. Mixed with carbonate of ammonia it is sold as smelling salts.

To its powerful corrosive action must be added the very injurious effect on the lung of its vapour, which gives rise to a broncho-pneumonia septic in type.

Fatal dose.—1 dr. of the strong solution has caused death.

Treatment.—Harmless acid drinks such as diluted vinegar or lemon-juice or citric or tartaric acid should be given freely. Pain should be relieved by the free use of morphine hypodermically, and nourishment should be given not by the mouth but per rectum.

The *vomit* is very alkaline to litmus paper, and should be reserved for the special chemical tests to determine the alkali present.

CARBOLIC ACID

Carbolic acid (phenol) is commonly used as a disinfectant for domestic and surgical purposes. Allied preparations, such as creosote, cresol, etc., have a similar poisonous effect. Lysol is a combination of cresol with soap, and is similar in action to phenol.

Pure carbolic acid is a colourless crystalline solid, liquefying with one-tenth its weight of water, but the commercial acid sometimes known as "carbolic" is a black, thick liquid which becomes milky on mixing with water. It is sometimes stated that the higher homologues of phenol, such as cresol, are not poisonous. This is untrue; they may be less poisonous, but they are nevertheless dangerous poisons.

Carbolic acid and the allied substances have a powerful corrosive action causing necrosis of the tissues with which they come into contact, the superficial part of which has a whitish appearance, the deeper parts being dark red owing to resulting hæmorrhage into them. This acid is one of the poisons most frequently used by suicides, and owing to its common

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employment for domestic purposes accidental poisoning by it often occurs.

The **symptoms** caused by carbolic acid are essentially those of corrosive poisoning, but owing to its local anæsthetic action vomiting may be absent and pain may be less pronounced. If death does not result from shock, the profound effect of the poison on the nervous system causes paralysis of the respiratory and cardiac centres, with rapid feeble pulse and stertorous breathing. Convulsions sometimes occur. In severe cases coma develops, and is usually followed by death.

The **urine** is either completely suppressed or small quantities may be passed containing oxidized products of phenol, such as hydroquinone, which on exposure to air give it a dark brown appearance.

Fatal dose.—1 dr. taken by the mouth has caused death in twelve hours. Death has also resulted from the absorption of phenol by the skin, and from rectal injections of the drug in solution; it occurs usually in from three to four hours after the taking of a large dose.

Treatment.—The stomach should be washed out by means of a soft stomach-tube with diluted saccharated lime-water or fresh lime-water; by this means the phenol is converted into calcium phenate, which is not poisonous.

Sodium and magnesium sulphate in solution have been recommended as antidotes to carbolic acid; it is extremely doubtful, however, if the conversion of phenol into sulphocarbonates occurs by contact with the inorganic sulphates in solution at ordinary temperatures. These sulphates, therefore, are not recommended as antidotes.

Olive oil has been used in phenol poisoning. It may be an advantage to introduce into the stomach $\frac{1}{2}$ –1 pint of olive oil, and after a few minutes to withdraw it by means of the soft stomach-tube, the stomach being then washed out with lime-water or weak sodium bicarbonate solution. Olive oil dissolves phenol, and so may remove it from the stomach-wall. Unless, however, it is removed from the stomach the phenol will be absorbed with it on its passage into the intestines.

General treatment, such as warmth, stimulants, rectal feeding, etc., is necessary to counteract the special symptoms arising.

Tests.—The vomit, stomach washings, and urine should be carefully reserved for analysis.

A delicate test is the white precipitate of tribrom-phenol that is given by the addition of bromine water to the liquid to be tested,

previously slightly acidified with dilute hydrochloric acid.

2. IRRITANT POISONS

The more important irritant poisons will be considered under this heading, but it must be remembered that many of the general poisons, e.g. savin and cantharides, have an irritant action on the stomach and intestines in addition to their special action on important organs and nerve centres.

Oxalic acid ($\text{H}_2\text{C}_2\text{O}_4 \cdot 2\text{H}_2\text{O}$) occurs in colourless crystals resembling Epsom salts and is very soluble in water. **Salt of sorrel** or **salt of lemon** is the quadrioxalate of potash ($\text{KH}_2\text{C}_2\text{O}_4 \cdot \text{H}_2\text{C}_2\text{O}_4 \cdot 2\text{H}_2\text{O}$); it is a white powder very soluble in water. Both of these substances are commonly used for domestic purposes, e.g. for cleaning straw hats, removing ink-stains, cleaning brasses, etc. They are frequently the cause of accidental and suicidal poisoning.

Symptoms.—When oxalic acid is swallowed in solution the typical symptoms of irritant poisoning are set up; in addition the poison, when absorbed, has a profound depressing action on the heart and nervous system. An acid burning taste is experienced and pain occurs which is referred from the throat, œsophagus, stomach, and later possibly the intestines. Vomiting is common, the vomit being very acid, giving the tests for oxalic acid, and often containing blood. Unless immediate treatment is adopted, collapse speedily supervenes, the patient becoming cold, pale, and faint, with a rapid feeble pulse, and being in danger of death from syncope.

In some cases nervous symptoms such as tinglings and numbness, muscular spasms, convulsions, delirium, and coma occur; but these are uncommon.

Owing to the rapid absorption of the poison, death is likely to take place rapidly, say within an hour, but it may be delayed.

Fatal dose.—60 gr. killed a boy of 16 in eight hours.

Treatment.—Fresh lime-water or, better, the saccharated lime-water, which is fifteen times as strong, should be given in large quantities mixed with calcium carbonate in the form of chalk or whiting. Chalk and whiting are useful antidotes, but, being insoluble, do not act quickly enough alone. Ceiling plaster—a mixture of calcium carbonate and sulphate—may be given, made into a cream with water.

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Since oxalic acid has only slight corrosive action the stomach should be washed out immediately if no antidote is at hand.

It is best to give the antidote freely if immediately available, after a few minutes to wash out the stomach thoroughly with a soft stomach-tube and funnel, and finally to introduce a pint of lime-water made into a thin cream with an ounce of chalk, leaving this mixture in the stomach.

Stimulant treatment is necessary to counteract the general effects of the poison; thus, normal saline with brandy (an ounce of the latter to the pint of saline) should be given freely by the rectum. Strychnine $\frac{1}{10}$ gr. and digitalin $\frac{1}{100}$ gr. are injected hypodermically, and repeated if necessary. Subcutaneous or intravenous saline should be employed when collapse is pronounced. In severe cases oxygen bubbled through absolute alcohol should also be utilized.

The *post-mortem signs* depend on the strength of the solution. When the solution is strong, signs of corrosive action on the mouth, throat, gullet, and stomach may be seen, the stomach showing areas of blackening and reddening around the destroyed mucous membrane. When the poison is taken in diluted form general redness of the stomach is present, the mucous membrane being intact but showing often petechial hæmorrhages; the duodenum and upper part of the intestine are involved if the poison has passed beyond the stomach.

The *vomit* should be made alkaline with ammonia, filtered, and to the filtrate should be added acetic acid in slight excess and calcium chloride solution, when a white precipitate of calcium oxalate occurs.

Arsenic is the most important of the irritant poisons, and, owing to the almost tasteless property of many of its compounds and preparations, it is perhaps the poison most commonly used for homicidal purposes.

White arsenic (i.e. arsenious acid, or arsenious anhydride, As_2O_3) is the most important of the compounds of arsenic. It occurs in the form of a white powder, or in lumps of a glass-like or porcelain-like appearance (vitreous arsenic). The powdered form resembles powdered sugar or flour, and when mixed with solid food is tasteless.

It is only slightly soluble in cold water, an ounce of cold water dissolving from half to one grain, but in boiling water it is twelve times more soluble, 6-12 gr. dissolving in an ounce.

Alkaline solutions readily dissolve arsenic.

If white arsenic is sold to the public the law requires that it be mixed with soot or indigo to colour it. It is used in the composition of sheep dip, arsenical soap, rat poisons, etc.

Metallic arsenic is a black powder, and is very poisonous; it is used for killing flies.

Copper arsenite (Scheele's green) is bright green in colour and was formerly used for colouring wallpaper, toys, floorcloth, fabrics, etc. Its use for such purposes has fortunately been abolished.

Arsenious sulphide (orpiment) is a yellow powder known as king's yellow.

Arsenic acid, in the form of its potassium and sodium salts, which are white, crystalline, and soluble in water, is used as a fly poison and in the manufacture of aniline dyes.

Injurious effects such as local skin eruptions are sometimes caused by the action of an arsenical compound present in the dye of stockings, etc.

Arsenuretted hydrogen (arsine) is a very poisonous gas. It is produced when hydrogen is generated in the presence of a compound of arsenic.

Salvarsan and the many similar derivatives of arsenobenzol which are extensively used in the treatment of syphilis and other diseases may give rise to fatal poisoning.

Sodium arsenite, in the form of solutions of arsenic in caustic soda or sodium carbonate, is commonly used for the preparation of fly-papers, weed-killer, preservative for wood, arsenical sprays for fruit trees, etc. Weed-killer and some of the so-called preservatives for wood contain as much as 20-40 per cent. of arsenic in solution and are intensely poisonous. Both weed-killer and the arsenic obtained from fly-papers have been used for homicidal purposes.

Arsenic used to have a reputation as a cosmetic and was added to face powders, skin lotions, etc., but now is practically never used for such purposes.

Arsenic in food.—Accidental contamination of food with arsenic or its preparations, such as weed-killer, has occurred. Arsenical pigments have been used for colouring sweets and cakes, with fatal results.

A serious epidemic occurred in 1900 due to the contamination of commercial glucose by arsenic. Sulphuric acid prepared from pyrites and containing a considerable amount of arsenic had been used in the process of the conversion of starch into commercial glucose, so that the latter became impregnated with arsenic. The

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glucose was used in the manufacture of beer, and so produced an epidemic in Manchester and the North of England. The cause of this epidemic was investigated by a Royal Commission, and the amount of arsenic permitted in foodstuffs was limited to $\frac{1}{100}$ gr. per gallon for liquids and per pound for solids.

The varieties of arsenical poisoning are as follows :—

(1) **Acute arsenical poisoning.**—When the poison is well diluted or mixed with food no taste or pain in the mouth or throat is experienced. The **symptoms** begin within an hour if the stomach is empty, but may be delayed if the stomach is full; also if the poison is in the solid state there will be further delay. A burning pain ensues in the epigastric region, and nausea and vomiting usually follow. As the poison passes on to the intestine, abdominal pain of a gripping or colicky type occurs, usually with diarrhoea. The continued vomiting and diarrhoea cause exhaustion, faintness, and collapse. Cramps in the legs may be experienced but are not a constant symptom. In a severe case restlessness, stupor, and coma develop, and death follows shortly. The vomit will contain any food that was in the stomach, often accompanied by much mucus. Bile is usually present, and sometimes there are streaks of blood. The stools are watery and may contain flakes of mucus. Death in an acute case may occur within twenty-four hours, or may be delayed for three days or more.

When several repeated doses are taken, so that the symptoms are protracted over several days, some of the symptoms of chronic arsenical poisoning may develop.

Fatal dose.—2 gr. of arsenic have caused death in a woman, and this is accepted as a possible fatal dose.

Post-mortem signs.—The signs of gastro-intestinal inflammation will be present. The stomach contents usually include much mucus, which may be bloodstained. The mucous membrane is swollen, red, and congested, and petechiæ are usually well marked. The redness is most obvious on the summits of the rugæ.

When the arsenic has been taken in solid form, white or pigmented particles may be seen on the mucous membrane of the stomach. The duodenum shows pronounced redness and congestion, and petechiæ may be present; there is usually yellow staining from altered bile. The small intestine may show similar

changes to those in the duodenum, but these diminish on passing downwards. There may be cloudy swelling of the liver, kidneys, and other organs. In fatal cases the arsenic absorbed into the tissues has a preservative action and tends to delay putrefaction.

Treatment.—The stomach should be washed out, and afterwards, as an antidote, freshly precipitated ferric hydrate may be given; this is prepared by adding to half a tumblerful of water half an ounce of tincture of perchloride of iron and sodium carbonate solution till the mixture is distinctly alkaline. Pain is relieved by hypodermic injection of morphine, demulcent drinks are given, and collapse calls for the usual stimulant treatment.

(2) **Chronic arsenical poisoning.**—In this condition the gastro-intestinal symptoms—nausea, abdominal pain, vomiting, and diarrhoea—are not prominent and may even be absent. The tongue is often covered with a silvery white fur. General malaise, anorexia, and anæmia are usually present.

Irritation of the throat and huskiness of the voice arise from the pharyngitis and laryngitis set up. Conjunctivitis may occur with redness and swelling of the eyelids. Skin affections such as erythema, herpes, pigmentation, and erythromelalgia may be produced. The pigmentation is a darkish discoloration of the skin commencing round the neck and spreading all over the body. Usually little white areas about $\frac{1}{4}$ in. in diameter are present over the affected parts. The mucous membranes are not affected, and the folds of skin, such as the axillæ and groins, are not specially attacked, these points serving to distinguish the condition from the pigmentation of Addison's disease. Thickening of the epidermis of the soles and palms and irregular thickening of the nails are present in long-standing cases.

Symptoms of multiple neuritis are likely to develop, affecting both the upper and the lower extremities. (See Arsenical Neuritis, under MULTIPLE NEURITIS.)

Long-continued poisoning causes great anæmia, progressive wasting, and heart weakness, death resulting from exhaustion and cardiac failure.

The urine, the fæces, the distal portions of the hair, and the nails will contain arsenic; its detection will serve to confirm the diagnosis during life.

Treatment consists in preventing the absorption of arsenic in any way, and in stimulant and eliminative measures.

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It must be remembered that in cases of suspected arsenical poisoning the diagnosis can always be made with certainty by an analysis of the urine, vomit, and feces; these should always be taken for examination, the tests being made by an expert toxicologist.

(3) **Arseniuretted hydrogen poisoning.**—Arseniuretted hydrogen is a very powerful poison, and cases of poisoning occur when hydrogen containing the gas is inhaled. Cases have also occurred when ferro-silicon, a substance used for hardening steel, has come into contact with water, for in these circumstances a mixture of arseniuretted and phosphoretted hydrogen is evolved. Arseniuretted hydrogen has a very toxic action on the liver and kidneys, and is also a blood poison.

Symptoms.—Malaise, headache, dizziness, and shivering appear within a few hours; vomiting follows, and jaundice, hæmaturia, and anæmia develop. Delirium, stupor, and coma precede death, the symptoms being exactly similar to those of icterus gravis.

Post-mortem examination shows pronounced degenerative changes in the cells of the liver and kidneys, and numerous petechiæ on the mucous and serous membranes.

(4) **Poisoning by salvarsan and arsenobenzol derivatives.**—The symptoms of this form of poisoning are described in detail under SYPHILIS. Treatment consists in the administration of alkalis by the mouth and bowel, and of saline per rectum. The prognosis is very grave.

Antimony.—The common preparation, potassium antimony tartrate, known as tartar emetic, is a white crystalline substance fairly soluble in water. It causes symptoms of gastro-enteritis very like those of acute arsenical poisoning.

Fatal dose.—3 gr. have caused death in an adult healthy woman; $\frac{1}{2}$ gr. has caused death in a child.

Tartar emetic has been used on several occasions for homicidal purposes, owing to the ease with which it may be administered without detection. Usually in such cases the symptoms have been mistaken for those of a natural disease.

Treatment.—Wash out the stomach. Give 1 dr. of tannic acid in solution by the mouth and adopt stimulant and eliminative treatment.

Antimony chloride (butter of antimony) is commonly extant as a solution, free hydrochloric acid being also present; the liquid is dark brown in colour. It causes symptoms similar to those of hydrochloric acid and

antimony combined. The treatment is similar to that for tartar emetic poisoning, but sodium bicarbonate should be given freely with the tannic acid.

In large doses in solution, metallic poisons such as those next considered—zinc, copper, barium, potassium bichromate, and silver nitrate—cause acute irritant poisoning like that of acute arsenical poisoning, except that often constipation rather than diarrhœa is present.

Zinc.—*Zinc sulphate* (white vitriol) occurs in the form of white soluble crystals exactly like Epsom salts. *Zinc chloride* has a corrosive as well as an irritant action; its solution is used as a disinfectant (Burnett's fluid).

Copper.—*Copper sulphate* (blue vitriol) exists as blue crystals soluble in water and is used for agricultural and industrial purposes. *Copper acetate* (verdigris) is a basic acetate.

Barium.—The *chloride* and *nitrate* are white crystals soluble in water. The *sulphate* is insoluble and is used in X-ray work for test meals.

Fatal results have ensued from the use of *barium carbonate* in mistake for the sulphate; the carbonate is used extensively in India for poisoning rats.

Barium salts in solution, in addition to acting as irritants, are powerful nerve-poisons.

Potassium bichromate is extensively used in the arts and for industrial purposes. It is a powerful irritant poison and also causes nephritis. In fatal cases the stomach and intestines show olive-green staining.

Silver nitrate in strong solutions is a corrosive poison, in weak solutions is an irritant.

The treatment of cases of acute metallic poisoning consists in washing out the stomach thoroughly and then giving freely by the mouth egg-albumen and bicarbonate of soda, also saline aperients, to which should be added, in the case of barium poisoning, sodium sulphate, and in silver poisoning, sodium chloride.

Mercury.—*Acute mercurial poisoning* is usually due to the taking of mercuric chloride (corrosive sublimate), or of the biniodide, both of which are used extensively as disinfectants; they are often taken in tablet form.

Symptoms of acute irritant poisoning occur and may clear up quickly after treatment.

Dangerous complications, however, must be expected, and comprise (1) suppression of urine, which develops in three to five days

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from taking the poison and is followed by death from uræmia; and (2) ulcerative colitis, which may be deferred for a week or more, but is a very dangerous complication.

Treatment.—The stomach should be washed out with warm water, and egg-white be given freely by the mouth. When suppression of urine supervenes, alkaline drinks, electric-light or hot-air baths should be given, with hot applications over the kidney. If these measures fail, operation on the kidney, such as decapsulation or incision, may be tried to promote the renal secretion. Such operations have been done with success.

Chronic mercurial poisoning may follow the administration of calomel or other mercurial preparations medicinally, or result from absorption of compounds of mercury, as occurs in workers exposed to nitrate of mercury (used in the preparation of felt hats), vermilion (sulphide of mercury), etc. Stomatitis associated with severe pyorrhœa and very offensive breath is a usual symptom, and with it appear anæmia, cachexia, tremors, and marked nervous symptoms, and the patient often relapses into a condition simulating pernicious anæmia.

It must be remembered that mercurial poisoning may be caused by vaginal or uterine douches containing mercurial preparations.

The **treatment** of chronic mercurial poisoning consists in the giving of saline aperients to promote free excretion by the bowel, and antiseptic treatment of the stomatitis. The anæmia is treated by the administration of iron and by attention to the nutrition and hygienic condition of the patient.

Lead.—*Acute* lead poisoning is very rare and may result from the taking of any soluble lead salt such as acetate of lead (sugar of lead).

The **treatment** is on the lines laid down for acute metallic poisoning, the antidote being sulphate of soda, or sulphate of magnesia; both of these form the harmless insoluble lead sulphate.

Chronic lead poisoning occurs in workers in lead such as those engaged in the manufacture of white lead or other lead compounds. Painters, plumbers, compositors, and file cutters are among those exposed to chronic lead-poisoning. The **symptoms** are anæmia, cachexia, intestinal colic, and renal symptoms due to chronic interstitial nephritis. A blue line is seen on the gums if the teeth are not kept clean, and is due to the deposition of lead sulphide in the gum near its borders. Joint-

pains are common, and chronic gout (poor man's gout) occurs.

Arterio-sclerosis is one of the most constant and most serious symptoms of chronic lead poisoning, and may be followed by its usual effects.

Neuritis, another important feature, usually affects the upper extremities. Wrist-drop due to musculo-spinal paralysis is a common symptom, but the supinator longus muscle is often unaffected. (*See also* Lead Neuritis, under MULTIPLE NEURITIS.)

Chronic lead poisoning is one of the most dangerous of diseases, and is unfortunately common among workers exposed to its risks.

The **treatment** consists in complete removal from all possibility of further absorption of the poison and in the free administration of sulphate of soda so that elimination by the bowel may be accelerated. Potassium iodide is of value for the arterio-sclerosis consequent on the condition, but it is doubtful if it increases the elimination of lead from the system.

Diachylon or lead plaster has often been taken for the purpose of causing abortion. If it succeeds in its purpose it generally also causes the death of the person taking it. The final symptoms, in addition to those already described for chronic lead-poisoning, are usually cerebral in type (lead encephalopathy); they comprise convulsions, stupor, delirium, and coma, terminating in death.

Bromine and **iodine** are irritant poisons, and should be treated by the administration of sodium bicarbonate, 2 dr. in solution in a tumblerful of water. Ammonia should also be inhaled. The stomach should be washed out.

Bromides taken internally are apt to cause the development of an acneiform eruption on the body, and in large doses may give rise to severe nervous depression, ataxy, loss of knee-jerks, and even fatal coma. (*See also* DRUG ERUPTIONS.)

Iodides taken internally commonly set up coryza, running from the eyes and nose, irritation of the throat, aching of the teeth, and an acneiform eruption which may be extensive and have one or more large lesions (*see* DRUG ERUPTIONS). Purpuric rashes sometimes also occur. Some persons exhibit a remarkable idiosyncrasy to iodine and iodides, and are quite unable to tolerate them either internally or externally.

Boric or **boracic acid** is extensively used as a food preservative. It is a gastro-intestinal

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irritant and may give rise to skin rashes such as erythema or a general urticaria; it is also a renal irritant, sometimes causing nephritis. Owing to its harmful effects, especially in infants and invalids, its use in milk as a preservative has been legally forbidden. For cream the limit has been fixed at 0.25 per cent., and for butter at 0.5 per cent.

3. GENERAL POISONS

Hydrocyanic acid, or prussic acid (B.P.), contains in solution 2 per cent. by weight of hydrogen cyanide. Scheele's acid contains from 4 to 5 per cent. **Potassium and sodium cyanide** are used in the arts and for industrial purposes. **Oil of bitter almonds** may contain hydrocyanic acid in appreciable amount.

Symptoms.—Hydrocyanic acid is one of the most rapid of poisons. It has a powerful action on the respiratory centre and quickly causes muscular paralysis. In a few seconds loss of muscular power and insensibility occur, the respirations become gasping, convulsions and incontinence of urine and faeces result, and death follows within five minutes.

With cyanide of potassium or sodium the symptoms may be somewhat delayed.

Fatal dose.—Half a drachm of the acid of the Pharmacopœia and 5 gr. of cyanide of potassium have proved fatal.

Treatment.—The onset of symptoms is so rapid that little time is available. As antidotes a dilute solution of peroxide of hydrogen, sanitas, or permanganate of potash should be given freely, since these oxidize the acid to harmless formic acid. It is said that 20 min. of a 10-per-cent. solution of sodium thiosulphate given hypodermically acts as an antidote.

If possible, the stomach should be washed out with a weak solution of permanganate of potash. If no stomach-tube is available, 30 gr. of ammonium carbonate should be given as an emetic. There should, however, be no delay in the adoption of artificial respiration and the administration of stimulants such as atropine and digitalis hypodermically and the inhalation of ammonia. Oxygen may be given, and faradization of the phrenic nerves resorted to if time permits.

Chloroform poisoning usually occurs from the administration of the drug as an anæsthetic. The symptoms may be immediate or delayed. Delayed chloroform-poisoning arises from the effect of the drug on the liver, and is dealt with under Liver Poisons (see below).

The immediate symptoms are usually due to

the effect of the poison on the respiratory centre, the breathing stopping while the heart continues to beat. In some cases heart failure occurs first, especially in status lymphaticus (see LYMPHATISM). In status lymphaticus there is found degeneration of the cardiac muscle, and in other conditions where this occurs (e.g. diphtheria) heart failure from chloroform administration is likely.

Treatment.—Immediate artificial respiration is all-important, and ammonia should be applied over the nostrils. Hypodermic injection of atropine $\frac{1}{10}$ gr., and of pituitary extract (1 c.c. of the pituitary extract solution, 20 per cent.), and the injection of spirit of ether (10 min.) over the præcordium are called for. Oxygen inhalation should be adopted without delay, and the bed or operating-table should be tilted so that the head inclines downwards. If all these remedies fail, opening of the abdomen and direct massage of the heart should be adopted.

Ether, ethyl chloride, and nitrous-oxide gas, when given as anæsthetics, may cause failure of the heart or respiration. The treatment of this condition should be on the same lines as for chloroform poisoning.

Chloroform or ether taken in poisonous doses by the mouth produces rapid insensibility and coma. The treatment consists in washing out the stomach without delay, and the introduction into the stomach of a saline aperient such as Epsom salts or Glauber's salts in solution. One pint of hot strong coffee should be given. Artificial respiration should be resorted to, and strychnine and digitalin given hypodermically.

Alcoholic poisoning is considered elsewhere (see ALCOHOLISM).

Liver poisons.—A very large group of poisons have a direct action on the liver, causing rapid loss of function; symptoms of auto-intoxication quickly result, and jaundice of a toxic type often occurs before death. These poisons may cause preliminary symptoms of gastrointestinal irritation, general malaise, etc., but the acute symptoms of auto-intoxication are very characteristic and run a rapid course, usually ending fatally. The pathological effect on the liver is that of a degeneration of the liver-cells, often associated with deposition of fat—the so-called fatty degeneration. The liver is enlarged, and yellow-coloured on section. The term "cirrhosis," which refers to the yellowness of the liver, was meant to apply to this condition, though it is often reserved for the fibrosed type of liver associated

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with alcoholic cirrhosis (Laennec's cirrhosis or hob-nailed liver). Should the patient survive the destructive effect of the poison on the liver, the damaged cells become replaced by fibrous tissue and a shrunken atrophic liver results, which is usually yellow or green from bile-staining.

Phosphorus, chloroform, and salvarsan (already referred to) are examples of liver poisons, as also are ether (*see* VOMITING, POSTANÆSTHETIC) and the vapour of tetrachlorethane, the latter of which was used in the manufacture of aeroplanes. Trinitrotoluene, dinitrobenzene, picric acid, and many of the compounds used in munition manufacture have a similar effect, and indeed the same is true of most of the aniline and benzene derivatives, such as toluylens-diamine and phenylhydrazine, etc.

The **symptoms** caused by these poisons are those of icterus gravis, for the condition is often associated with jaundice, which may be slight or deep; they are restlessness, irritability, often vomiting (the vomit being sometimes bloodstained), delirium, stupor, twitchings of muscles and possibly convulsions, Cheyne-Stokes breathing, coma, ending in death. These symptoms run a rapid course, lasting usually only two or three days. The rectal temperature is frequently raised shortly before death.

The **treatment** of this threatening condition is to administer alkalis freely by the mouth, e.g. sodium bicarbonate $\frac{1}{2}$ dr. in water or soda-water every hour, and per rectum normal saline containing 2 dr. of bicarbonate of soda to the pint as freely as it can be retained. Stimulants such as strychnine and digitalin should be injected. Hot coffee should be given by the mouth and normal saline may be injected subcutaneously or intravenously. The bowels should be made to act by a dose of calomel or saline aperient.

This state of auto-intoxication due to cessation of liver function is so rapid and fatal that there should be no delay in the adoption of the treatment advised, since in the early stages it may ward off further symptoms and save the life of the patient.

Organic compounds used as hypnotics.—These, if taken in excessive doses, act as poisons. The symptom which attracts attention is the deep coma produced. The time of onset of coma depends entirely on the rate of absorption of the poison; if the latter is insoluble, coma is delayed, if soluble its appear-

ance is rapid. Examples of this group are chloral, veronal and its derivatives, medinal, proponal, luminal, and dial, also sulphonal, trional, and tetronal. Any hypnotic drug, if taken in excessive quantity, acts as a coma-producing poison.

The symptoms of veronal and its derivatives deserve special mention. In addition to the deep coma which rapidly supervenes, there are usually a rise in temperature, marked cyanosis, cough, and definite signs of dullness with tubular breathing and moist sounds over one or more lobes of the lung, so that the signs closely resemble those of pneumonia. The diagnosis from that condition is made by the difference in onset, the absence of pain and rigor, also the absence of the characteristic tenacious rusty sputum. In fatal cases of veronal poisoning the lung is found in a state of intense cedema and congestion—the causes of the physical signs during life.

The **treatment** for poisoning by the above hypnotics consists in immediate washing out of the stomach, and administration of a pint of hot strong coffee and of a purgative such as an ounce of castor oil.

A most essential part of the treatment is the regular feeding of the patient while in the comatose state. This should be done by a nasal or stomach tube every six hours while the coma lasts. *In my opinion, absence of this precautionary measure is responsible for many of the deaths from coma-producing drugs.* Cardiac stimulants such as digitalin and strychnine should be injected hypodermically. In severe cases normal saline should be given by the mouth and subcutaneously, and oxygen administered.

VEGETABLE POISONS AND THEIR ALKALOIDS

This group of poisons is so large that it is impossible to deal with them all individually, and only a few of the more important members will be considered.

When the poison has been taken by the mouth the **immediate treatment** should consist in evacuation of the contents of the stomach by means of a soft tube and funnel, the stomach contents or vomit being carefully reserved for analysis. The stomach should then be washed out thoroughly with warm water; it is advantageous to tint this a decided red colour with permanganate of potash, and to continue the lavage until the liquid returned from the stomach has the colour of the permanganate. An aperient such as an ounce of castor oil

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should usually be given, and also a pint of hot coffee with milk.

Symptoms are treated by appropriate remedies as they arise. Should the breathing show any tendency to fail, artificial respiration must be resorted to. Should coma supervene and be of long duration, feeding by the nasal or stomach tube at intervals of four or six hours is most important.

Opium and morphine.—Opium is very largely used in medicine, and its various medicinal preparations are well known. Morphine, an alkaloid of opium, is present in it to the extent of about 10 per cent. in the form of meconate of morphine.

It must be remembered that children are exceedingly susceptible to the effects of opium and morphine, and that poisoning may easily arise from small doses, so that the greatest care is necessary in prescribing the drug and its preparations for children, only a very weak preparation, as the tinct. camphoræ composita, strength 1 in 240, being used.

Fatal doses.—4 min. of tincture of opium have killed a child of 9 months, and 1 min. has proved fatal to a child of 7 days. In adults 2 dr. of tincture of opium, 4 gr. of opium, and 1 gr. of morphine have each proved fatal.

Opium and its preparations, as already stated, are exceedingly toxic to patients with kidney or liver disease, in which the excretory power of the organ is diminished—e.g. chronic nephritis, cirrhosis of liver—and they should not be given in these conditions. In pneumonia and diseases where activity of the respiratory centre is all-important, opium and morphine should be used with the greatest caution.

The **symptoms** come on usually within an hour, and, after a short period of mental excitement, lassitude, stupor, and coma supervene. The respiratory centre is profoundly depressed, and the breathing becomes slow and shallow and afterwards may be stertorous. The pupils are pin-point, but may become dilated before death. The skin is cold and moist.

In morphine poisoning, in addition to the symptoms enumerated, vomiting sometimes occurs, and occasionally convulsions are marked. Death usually follows within twenty-four hours unless treatment is adopted.

Treatment consists in thoroughly washing out the stomach with water coloured with permanganate of potash, which, oxidizing the morphine, renders it innocuous.

The lavage should be repeated at two-hourly intervals twice or thrice, since the morphine is excreted into the stomach after absorption. Ammonia should be applied to the nostrils, and atropine injected hypodermically. By the mouth, hot strong coffee, and castor oil, should always be given promptly.

Artificial respiration should be continued as long as there is danger of failure of breathing.

Heroin (diacetyl morphine) and **dionin** (ethyl morphine) are alkaloids which may both cause symptoms of poisoning like those of morphine, and the treatment is similar.

Belladonna (deadly nightshade) and its active principle atropine act in large doses as deliriant poisons. Atropine is a powerful mydriatic, and paralyses the salivary and sweat-glands.

The **symptoms** of belladonna and atropine poisoning are dryness of the mouth and throat and of the skin, giddiness, wide dilatation of the pupils, excitability and delirium. Often there are a rise of temperature and an erythematous rash like that of scarlet fever. Stupor and coma intervene in severe cases, and death may result within twenty-four hours.

Stramonium, **hyoscyamus** (henbane), **hyoscyamine**, **hyoscine**, and **scopolamine** all produce symptoms similar to atropine poisoning, the wide dilatation of the pupil due to complete paralysis of the sphincter to light and accommodation being a prominent feature.

Treatment is on similar lines to that for other vegetable poisons. Pilocarpine nitrate $\frac{1}{4}$ gr. should also be given hypodermically, and repeated every four hours until the skin becomes moist. If there is much delirium, morphine $\frac{1}{4}$ gr. should be given hypodermically and repeated.

Cocaine poisoning.—Cocaine, usually in the form of the hydrochloride, is commonly used as a poison. It may be taken by the mouth or given hypodermically. Sometimes it is employed in the form of a nasal or inhalation spray. When it is taken by the mouth, numbness and anaesthesia occur locally, and great excitement with tendency to violence is produced, so that the patient may destroy furniture, etc. Hallucinations and delirium quickly supervene, followed by coma. The pupils are dilated, the pulse is rapid and feeble, and sometimes convulsions occur.

Fatal cocaine poisoning has resulted on many occasions from injecting a solution into the urethra. Death has followed the injection of 20 min. of a 40-per-cent. solution. Probably in these circumstances there is very rapid

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absorption under pressure from a raw surface. In cases of this type convulsions and loss of consciousness occur, followed by death in a very few minutes.

The **treatment** is similar to that of morphine poisoning, except that, instead of atropine, digitalin $\frac{1}{100}$ gr. should be given hypodermically. Rectal saline, or saline subcutaneously or intravenously, is necessary in severe cases. Oxygen bubbled through alcohol is also a valuable remedy as a cardiac stimulant.

The **drug habit** is very likely to follow the use of morphine or cocaine hypodermically, and these drugs should be avoided as far as possible and only given under strict supervision. The patient should never on any account be allowed to administer them hypodermically to himself. The drug habit may also follow the use of opium, morphine, or cocaine when taken by the mouth; indeed, the use of almost any drug having an action on the nervous system, such as veronal, hyoscine, paraldehyde, etc., may be followed by the development of a drug habit which requires special treatment. (See DRUG HABIT.)

Nux vomica and strychnine.—Strychnine, the alkaloid of nux vomica, has a powerful action on the motor nerve-cells, causing in poisonous doses the rapid appearance of general convulsions which recur at short intervals, death resulting usually within a few hours.

Treatment.—Chloroform inhalation should be given and the stomach freely washed out while the patient is under the anæsthetic. Chloral hydrate 20 gr. and potassium bromide 60 gr. should also be given, and repeated if necessary. The chloroform inhalation should be continued while there is a tendency to recurrence of the convulsions.

Aconite and aconitine cause numbness locally, and numbness and anæsthesia may also occur in the limbs from absorption of the poison. Aconitine is a powerful cardiac and nerve poison. It acts quickly, causing collapse, rapid feeble pulse, convulsions, loss of power of the limbs, coma, and death.

Treatment is on the same lines as for morphine poisoning, and digitalin $\frac{1}{100}$ gr. should be given hypodermically and repeated as often as necessary.

Renal poisons.—Renal poisons such as **turpentine** act as gastro-intestinal irritants in addition to causing lumbar pain, hæmaturia, and suppression of urine. The same may be said of **cantharides**, which may be mentioned here, though not of vegetable origin.

Treatment.—The stomach must be washed out, a purge given, and everything possible done to induce the kidneys to resume their function. Hot bland drinks such as milk, barley-water, and lemon drink should be given freely. Citrate of potash and caffeine are of value. Hot fomentations and dry cupping over the loins are also helpful.

4. GASEOUS POISONS

Carbon-monoxide poisoning.—This occurs from coal gas, water gas, or exposure to suffocating fumes from fires, smoky grates, stoves, or lamps, also to fumes from geysers.

The **diagnosis** may be made at once by the clinical symptoms of drowsiness, collapse, and coma, and the cherry-red colour of the face and mucous membranes. An examination of the blood will make the diagnosis certain, since the presence of carbon-monoxide-hæmoglobin may readily be detected by the spectroscope and the solution of blood in water has a characteristic cherry-red colour quite different from that of normal blood.

Treatment.—Oxygen must be given freely and artificial respiration employed if necessary. Strychnine and digitalin should be administered hypodermically, brandy by the mouth, and warm saline either subcutaneously or by the bowel. For heart failure pituitary extract may be injected hypodermically if necessary.

Carbon-dioxide poisoning (carbonic acid poisoning) is met with in coal mines from choke damp or after-damp. Cellars of houses, wells, brewers' vats, lime-kilns, etc., frequently contain large quantities of carbonic-acid gas. The gas is produced in various combustion processes, and also occurs in air which has been rendered impure by respiration, as in excessive overcrowding.

Treatment consists in fresh air, rest, oxygen, artificial respiration if necessary, and the employment of warmth and stimulants where required.

Sulphuretted hydrogen poisoning results from contamination of the air with sewer gas, as from the gases evolved when iron slag becomes moistened with water. The gas is frequently produced in various chemical processes. The symptoms are catching of the breath due to the irritant effects of the gas, cyanosis, dyspnoea, slow respiration, feeble pulse, headache, sickness, and muscular prostration; and in severe cases, unconsciousness, convulsions, or coma.

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Treatment.—Oxygen and stimulants must be administered, and the patient must have plenty of fresh air. Rest in bed for a few days is desirable, and the anæmia should be treated by iron and arsenic and special diet.

Chlorine.—This gas, which even in minute proportion in the air acts as a powerful irritant to the lungs, causes an acute bronchial catarrh and pulmonary congestion, which may be followed by broncho-pneumonia and other serious complications. Intense cyanosis and dyspnœa occur quickly, and may be accompanied by severe spasm of the glottis, which is often combined with some œdema. So difficult may respiration become that a bronchus may be perforated as a result of the convulsive respiratory movements, and the escaping air into the mediastinum may cause general subcutaneous emphysema.

This gas was extensively used during the War, and by its immediate effects caused a large number of deaths. The after-effects of chlorine poisoning may last for months or years, the patient being liable to paroxysmal attacks of dyspnœa, in many cases functional in nature. (See also GAS POISONING OF WAR, AFTER-EFFECTS OF.)

Treatment.—Prevention is ensured by the wearing of a suitable protective helmet or other apparatus. Sodium thiosulphate and sodium plenate have the property of absorbing chlorine effectually, and were used for this purpose in the field. Inhalation of an alcoholic solution of ammonia, if following immediately upon the inspiration of the gas, gives much relief. For the severe symptoms a steam kettle into which a little ammonium carbonate has been introduced is of value. Oxygen should be given freely; this is best done by means of a special apparatus such as the mask devised by Haldane.

5. POISONOUS FOODS

Poisonous fungi are sometimes eaten in mistake for edible mushrooms, and dangerous or even fatal results may follow. Thus the fly fungus, *Amanita muscaria*, contains the highly poisonous alkaloid muscarine; another fungus, *Amanita phalloides*, contains phallin or amanita, a powerful toxin, as its active principle; while *Helvella esculenta* owes its toxic properties to helvellic acid. The immediate symptoms caused by poisonous fungi are those of acute gastro-enteritis, together with collapse from the effect of the poison on the nerve-centres; usually the liver suffers severely, and death

from auto-intoxication may occur after a few days owing to the liver and possibly also the kidneys ceasing to function.

Poisonous fish.—Certain fish, such as those of the species *Tetrodon*, found in Japanese waters, are normally poisonous, and the same applies to mussels when grown under unhealthy conditions. The symptoms caused by these poisonous foods are those of acute gastro-enteritis with severe nervous disturbance and collapse.

Ergotism.—The grains of cereals attacked by the ergot fungus (*Claviceps purpurea*) are poisonous and give rise to a series of symptoms known as ergotism. Two types of this condition are known—(a) the gangrenous type, in which gangrene of the toes or fingers, and occasionally of the ears or end of the nose, occurs; (b) the nervous type, in which sclerosis of the posterior columns of the spinal cord, or degenerative changes in the nerve-cells of the brain, cause characteristic nervous symptoms.

Lathyrismus.—The addition of the meal of certain vetches (*Lathyrus sativus* or *L. cicera*) to the flour of cereals in the preparation of bread may give rise to serious nervous symptoms of the type of spastic paraplegia, the toxic substance derived from the vetches causing a slow sclerosis of the spinal cord. Outbreaks of lathyrismus have occurred in Italy, Algiers, and India.

Potato poisoning.—Under certain conditions potatoes may contain a dangerous amount of the poisonous alkaloid solanine; this is produced by the growth of certain forms of bacteria in the tubers, which is likely to occur if, owing to improper storage, the potatoes have sprouted. The symptoms of solanine poisoning are those of acute gastro-enteritis associated with nervous prostration and collapse; they resemble those caused by poisonous fungi.

Chemical poisoning.—Chemical toxins may accidentally contaminate foods and give rise to the symptoms characteristic of chemical poisoning, as described above. The following are examples:

Tinned foods.—These may be contaminated by poisons derived from the tin. It is a rare form of poisoning, and is only likely to occur when the contents of the tin are liquid, such as fruit juice, liquefied jelly, or soups. If the tin is sound and care is taken in the canning and in the process of soldering so that the solder does not come in contact with the contents, the risk of poisoning is negligible. Cases of poisoning have occasionally occurred from the tin, or the

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lead in the solder, having been dissolved by the contents of the tin. Another form of chemical poisoning has already been described under Arsenic.

Drinking-water may be contaminated by lead or copper and give rise to chronic poisoning.

Food preservatives, such as boric acid, formalin, salicylic acid, etc., may give rise to illness from their toxic effects.

Specific diseases may be communicated by the accidental contamination of food with pathogenic organisms. Thus, typhoid or paratyphoid fever, scarlet fever, diphtheria, cholera, amœbic or bacillary dysentery may be caused by infection of milk, oysters, and other food with the organisms of those diseases. A very common cause of these forms of food poisoning is the handling of food by apparently healthy persons who harbour the specific germs of a particular disease (see CARRIERS OF INFECTION).

Diseases in animals communicable to man may arise from the consumption of food derived from a diseased animal. Thus the flesh of an animal which has died from *anthrax* may give rise to this disease in man. Tuberculosis of the mammary glands in the cow leads to the presence of tubercle bacilli in milk, the consumption of which may give rise to human *tuberculosis*. The flesh of animals suffering from *actinomycosis* may cause this disease in man.

Parasitic diseases such as trichinelliasis (trichinosis) and tape-worm infections arise from the consumption of the flesh of animals suffering from such infections. These types of food poisoning are described in the articles on TRICHINELLIASIS, INTESTINAL WORMS, etc.

BACTERIAL FOOD POISONING (*syn.* PTOMAINE POISONING)

This is the form of poisoning caused by the contamination of food with certain forms of bacteria which by their toxic substances set up symptoms. There may also be an actual bacterial infection should the organisms not be killed in the process of cooking.

Since bacterial contamination finds a most suitable culture medium in foods rich in protein, this type of food poisoning usually follows the consumption of meat, fish, or milk.

Etiology.—It was formerly thought that food poisoning was caused by the presence in food of poisonous alkaloidal substances called *ptomaines* produced by putrefactive changes, of which *neurin* and *mydalein* are examples: it is now known that *ptomaines* are very rarely, if

ever, the cause of food poisoning, but that the usual cause is bacterial contamination. It is possible that in the consumption of food far advanced in putrefaction, such as high game, *ptomaines* may play a part in the causation of toxic symptoms, but such cases are quite uncommon.

Bacteriology.—The organisms which cause food poisoning usually belong to the colony-typhoid-dysentery group. The most frequent of these is the *Bacillus enteritidis* of Gärtner, but organisms of the paratyphoid group and those allied to the *Bacillus coli communis* are commonly found. In some cases organisms of the bacillary-dysentery type are found. The *Bacillus botulinus*, an anaerobic organism, has been occasionally found in sausage poisoning (botulism).

Food poisoning may result from any contamination with a bacterial poison, and in some cases the symptoms have been thought to be due to a streptococcal infection, and occasionally to the proteus bacillus.

Predisposing causes.—Contaminated food has a much more serious effect on persons suffering from pre-existing gastro-intestinal disease such as colitis or enteritis. Dysentery and post-dysenteric colitis have a like effect.

Starvation or malnutrition increases the susceptibility to bacterial food poisoning, and emptiness of the stomach at the time of the taking of contaminated food is likely to lead to the earlier onset and greater severity of the symptoms produced.

The above considerations help to explain the fact that sometimes, when several persons partake of the same unwholesome food, some may suffer from serious symptoms while others may only exhibit very slight symptoms or may even be unaffected.

Age.—Young children are much more susceptible than older persons.

Climate.—In hot climates bacterial food contamination is likely to be followed by a very rapid development of bacterial poisons.

General hygienic conditions.—Dust, flies, dirt and insanitary conditions generally are likely to favour the contamination of food with bacteria. Careful storage of food in proper safes, where it can be kept cool and free from contamination, is most important.

Morbid anatomy.—The post-mortem signs are those of an acute gastro-enteritis, with evidence of a general bacterial infection or toxæmia.

The tissues of the body may be shrunken:

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from drainage of fluid when the diarrhoea has been very severe. Rigor mortis sets in early.

The *stomach* may show some redness from congestion and, especially when vomiting has been severe, there may be small submucous hæmorrhages. Much mucus is generally present, but the stomach contents are usually free from blood. The inflammatory signs are, as a rule, more marked in the *small intestine*—an important distinction from chemical poisoning such as that caused by arsenic; the mucous membrane is swollen and congested, and the lymphoid follicles and Peyer's patches often show much swelling and congestion. The signs are frequently more pronounced in the ileum than in the upper part of the small intestine. The mucous membrane of the *large intestine* may be swollen and congested and the lymphoid follicles much enlarged. The contents are liquid and may contain much mucus. Small submucous hæmorrhages may occur in any part of the intestinal tract. The *spleen* is congested and soft, and in cases where infection with living bacteria has occurred it may be enlarged. The *liver* and *kidneys* may show cloudy swelling or fatty degenerative changes.

It is important to remember that in fatal cases of food poisoning the macroscopic morbid changes found post mortem may be very slight.

Symptoms.—The period which elapses between the taking of the contaminated food and the onset of symptoms may vary from a few hours to three or four days. The greater the amount of preformed bacterial toxins in the food, the earlier will be the appearance of symptoms. The latent period is influenced by other conditions also. Thus, if the poisonous food is taken on an empty stomach the symptoms will arise earlier. Again, if the infected food is taken alone the symptoms will occur earlier than if it is taken with a considerable amount of wholesome food. In the Welbeck epidemic, investigated by Ballard, the latent period was accurately determined in a number of cases. In 5 it was 12 hours or less; in 34 it was 12 to 36 hours; in 8 it was 36 to 48 hours.

The symptoms are of sudden onset, and are usually those of gastro-enteritis. The tongue is furred, and there is severe abdominal pain with a rise of temperature. Vomiting and purging are often present, associated in severe cases with considerable collapse and heart weakness. The stools are liquid and very offensive, and sometimes contain mucus and blood. In some cases the attack is ushered in by a rigor. In severe cases cramps occur in

the calves of the legs, and the patient may become cold and blue owing to the feeble circulation consequent on a state of extreme collapse. Erythematous or urticarial, or in bad cases purpuric, rashes may appear. Severe cases often present the symptoms of bacterial infection of the blood (septicæmia), and such complications as pneumonia are then likely to occur.

In some attacks of ptomaine poisoning, especially those due to contaminated fish, the toxins appear to act especially on the blood-vessels, producing erythema and urticaria with considerable swelling of the skin; these symptoms are associated with fever and a furred tongue, though there may be little or no diarrhoea or vomiting.

Meat poisoning is the commonest form of food poisoning. The meat may have been contaminated during the cutting-up after the slaughtering, or in the process of cooking, or later owing to lack of care in storage.

Sausages.—Many epidemics of poisoning have occurred from infected sausages. This can be readily understood, since the process of mincing which the meat undergoes renders it specially liable to contamination. Moreover, in sausages it is easy for unwholesome meat to be used with little possibility of detection. The fact that sausages are made with raw or only partly cooked meat, and that they are frequently eaten merely smoked, with little or no cooking, further adds to the risk.

The types of illness are similar to those described above, but in the sausage poisoning associated with the *Bacillus botulinus* (see BOTULISM), vomiting, diarrhoea, and abdominal pain are frequently absent, the symptoms in such cases being almost entirely of the paralytic type.

Tinned meat may be contaminated by poisonous bacteria before tinning, and though the process of canning may, by the heat applied, kill the organisms, the toxins may not be destroyed, and will give rise to symptoms of poisoning when the food is eaten.

Fish.—Numerous epidemics of food poisoning have been due to the eating of fish contaminated with bacteria of the types already described, and the resulting symptoms have been similar in character. It should be emphasized that the cooking of fish may not entirely destroy any toxins that are present.

Shellfish are frequently grown in waters polluted by sewage. Epidemics have been caused by mussels, oysters, lobsters, crabs, etc.,

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Usually the fish appeared to be perfectly fresh, with no objectionable taste or smell.

Tinned fish, like tinned meat, may give rise to poisoning from infection with bacteria before canning. It is important to remember that tinned fish, even if wholesome at the time the tins are opened, is then very liable to bacterial contamination. It should never be kept after the day of opening.

Vegetables and fruit, if contaminated by the bacteria described, may cause outbreaks of food poisoning. Since, however, the bacteria find vegetables an unsuitable medium for growth, such outbreaks are uncommon.

Milk.—If milk is infected with the organisms in question, all that is necessary to their growth is a suitable temperature; if this is present, the milk speedily becomes a dangerous article of food. Cases of poisoning by milk are therefore of much commoner occurrence in warm weather.

Complications and sequelæ.—An attack of bacterial food poisoning may be complicated by the development of a specific disease, such as enteric or paratyphoid fever, owing to the living organisms of this disease being also present in the contaminated food.

After an attack of bacterial food poisoning there is sometimes left a latent bacterial intestinal infection, so that the patient, in addition to being a carrier of disease, is liable to recurring attacks of enteritis from slight causes.

Course and prognosis.—In mild cases the symptoms clear up in a few days and the patient is completely restored to health. When there is an infection with *living* pathogenic organisms the illness may run a course similar to that of typhoid fever. Severe cases may present symptoms so acute as to resemble true cholera, and in these the prognosis is grave.

Diagnosis.—This is usually clear, from the sudden onset of typical symptoms not long after the taking of contaminated food. It cannot be too strongly emphasized that the symptoms of bacterial food poisoning are almost identical with those of poisoning by arsenic or antimony or other chemical irritant poison. An analysis of the vomit, feces, and urine for arsenic and irritant chemical poisons will render the diagnosis clear.

Treatment.—The importance of *prophylaxis* has already been indicated; the greatest care should be taken to avoid articles of food that are likely to be contaminated, and the thorough cooking of food whereby any living bacteria present are destroyed is a great safe-

guard. After cooking, rigid care should be taken in the storage of food.

General treatment.—Rest in bed is essential. The diet should consist of liquids such as water, albumen water, chicken broth, and whey. Milk is often best avoided, but peptonized or citrated milk (2 gr. of citrate of soda to the ounce of milk), diluted with an equal quantity of water, may be given. The feeds should consist of 3 or 4 oz. of liquid every two hours; one or two teaspoonfuls of brandy may be added with advantage. Hot applications to the abdomen are advisable. When there is much pain, or diarrhœa without collapse, the hypodermic injection of morphine is advisable. Colonic irrigations with 2 pints of normal saline are of value in cases with diarrhœa and slight collapse. If choleraic symptoms and severe collapse are present, normal or hypertonic saline should be given subcutaneously or intravenously. A mixture of bismuth and soda with hydrocyanic acid is valuable in relieving the gastric irritation; to control the diarrhœa, bismuth salicylate in 10- or 15-gr. doses is useful. As the symptoms subside the dietary is gradually increased.

The treatment of food poisoning should be accompanied by periodical bacteriological investigations of the stools. A case cannot be regarded as cured until three successive bacteriological examinations, made at intervals of two or three days, have given negative results.

W. H. WILLCOX.

POISONS, SALE OF (see POISONS AND POISONING).

POLIOENCEPHALITIS.—This term is usually restricted to inflammatory lesions of the grey matter of the brain-stem involving the nuclei of one or more of the cranial nerves. Two forms are described, polioencephalitis inferior being limited to the grey matter of the medulla and pons, and polioencephalitis superior affecting that of the midbrain. Polioencephalitis is, however, usually only part of a more extensive disease of the nervous system. The bulbar nuclei, and less frequently the mesencephalic, are often involved in acute poliomyelitis, in acute encephalitis, and in pyæmic conditions, while inflammatory lesions in the grey matter of the midbrain are the most characteristic features of encephalitis lethargica (q.v.).

One form, known as **acute hæmorrhagic superior polioencephalitis**, deserves special

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mention. It is an acute hæmorrhagic affection with little evidence of inflammation, which is more or less limited to the walls of the third ventricle and the grey matter around the aqueduct of Sylvius, but the lesions may extend to the floor of the fourth ventricle. It is most commonly associated with chronic alcoholism, but may be due to food poisoning (botulism), or to poisoning by sulphurous acid gas, and it occurs in the course of infectious diseases, as influenza. The onset is acute, with headache, vertigo, vomiting, increasing stupor or agitation, and paralysis of some or all of the ocular muscles, particularly of those supplied by the third nerves. Paralysis, alterations in the reflexes, or sensory disturbances in the limbs are rare, but tremor or ataxia is common. In both its pathology and symptoms it resembles lethargic encephalitis, but it does not occur in epidemic form and it is almost always fatal. Treatment has no effect on its course.

GORDON HOLMES.

POLIOMYELITIS, ACUTE. Etiology.—

Acute poliomyelitis has been observed on every continent, but appears to prevail in an epidemic form more frequently and severely in North America and Scandinavia than in other countries.

The great majority of cases occur in children under 5 years of age, and susceptibility to the disease diminishes progressively and somewhat rapidly to the end of the fourth decade, after which its incidence is extremely rare. The two sexes are equally affected in childhood. The seasonal incidence is related to the summer months; in this country the majority of acute cases are seen between June and October, when epidemics are liable to prevail. No other etiological factors are of importance; the disease attacks robust and healthy as frequently as delicate children.

Pathogenesis.—For many years it has been recognized that acute poliomyelitis was an infective disease and belonged to the category of the acute specific fevers, but it was not until 1909 that experimental inoculations of monkeys from the spinal cords of fatal cases of poliomyelitis were successfully made. In these animals a disease similar to that which affects the human race can be produced by inoculations into the brain, the peritoneal cavity, or the general circulation. The responsible virus can pass through the finest filters, and is resistant both to the action of glycerin and to considerable degrees of cold. It may be found in the

naso-pharyngeal mucous membrane of patients, and of persons who do not develop the disease but who have been in contact with its victims. The exact method of transmission is still obscure, but everything points to the guilt of "carriers" rather than of patients. The serum of patients and of monkeys who have survived an attack contains neutralizing principles which confer immunity to second attacks, and it can neutralize emulsions containing the active virus when added to them. The injection of an immune serum into monkeys affords little protection against subsequent infection.

In recent years Flexner and Noguchi have succeeded in cultivating a small globular organism averaging 0.15–0.3 of a micron in size. Monkeys have been successfully inoculated with subcultures, and typical poliomyelitis produced.

Morbid anatomy.—Post-mortem examination of an acute case reveals a hyperæmia of the cerebral and spinal meninges and a congested, sometimes hæmorrhagic, condition of the grey matter. If the cord is cut across, the grey matter stands out prominently on account of its vascular engorgement. Microscopically, the vessels of the leptomeninges are congested and an excess of lymphocytic cells is seen in the tissues around them. This cellular infiltration is usually greater on the antero-lateral than on the posterior surface of the cord. The presence of a meningeal reaction is suggested during life by a fairly constant lymphocytosis of the cerebro-spinal fluid when withdrawn by lumbar puncture during the acute stage of the illness.

The white columns do not show great changes, although scattered patches of disease may be observed, and there is often some neuroglial proliferation and cellular infiltration of the walls of vessels which penetrate them from the periphery of the cord; the cells lie chiefly in the lymphatic space between the media and adventitia and in the meshes of the latter. The grey matter, especially the anterior horns of the spinal cord, is most severely affected. In this tissue the vessels of all sizes are infiltrated by cells, and capillary hæmorrhages are by no means uncommon. In cases which have survived some days or weeks there is often evidence of thrombosis with consequent softening of the tissues. The grey matter itself is also the site of extensive cellular infiltration and of serous exudation.

Wherever the inflammatory changes are well

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marked the nerve-cells suffer in corresponding degree; this is the case not only in the anterior horns but in Clarke's column and in the posterior horns. Some cells preserve a more or less healthy appearance, others have a ghost-like resemblance to their former selves, and large numbers may be lost in the inflammatory exudation.

The spinal roots and posterior root ganglia often contain a certain amount of cellular infiltration, and the efferent fibres of the anterior roots, and sometimes those of the efferent cranial nerves, undergo secondary degeneration.

When patients have died many months or years after an acute attack, fibrotic scars deforming the grey matter may be found, or fluid-containing cavities in places where destruction has been complete. Large perencephalous cavities of this origin have been observed in the cerebral hemispheres.

The paralysed muscles undergo a simple atrophy, an increase of connective tissue taking the place of the degenerated muscle-fibres. Sometimes there is an increase, too, of fat, which gives the muscle a bulk out of proportion to its functional activity. The muscle-spindles remain intact.

The above description of changes in the spinal marrow is applicable to the brains of those cases of polioencephalitis which are due to the virus of poliomyelitis. It is in the more vascular grey matter of the encephalon that the most extensive inflammatory changes are found. It should be noted, however, that even when the clinical symptoms of a case of poliomyelitis suggest a disease confined to the spinal cord, the vessels of the brain-stem and sometimes of the cerebral hemispheres usually show the characteristic perivascular cellular infiltration. Speaking generally, the post-mortem evidences of inflammation are much more widespread than the clinical picture would lead one to expect.

Symptomatology. Early stage.—Poliomyelitis is a specific fever with a somewhat abrupt onset, following an incubation period of seven to fourteen days and running a course which is rarely longer than one or two weeks. In some cases the disturbance of health is so slight that the onset of paralysis in one or more limbs may appear without any obvious warning. In other cases the initial symptoms are so prominent that the presence of paralysis may escape notice until the fever has subsided and convalescence has begun. In

what may be called a typical instance a child in the best of health rather suddenly suffers from restlessness, anorexia, and fever which may reach a temperature of 100-104° F. There may be initial convulsions with vomiting, and it is not very uncommon at this stage to find catarrh of the naso-pharynx or of the gastro-intestinal tract. The usual diagnosis of influenza or a feverish chill is upset a day or two later by the supervention of paralysis in one or more limbs, with or without spontaneous pain, but nearly always accompanied by intense discomfort when any attempt is made to handle or manipulate a paralysed part. The paralysis may be widely distributed from the first, but it often begins in one or two limbs and spreads in the course of twenty-four or forty-eight hours to other parts of the body. At the end of a few days all constitutional disturbances pass off; the appetite is regained, the temperature descends to normal, and only the local results of the spinal inflammation remain manifest. At this stage a certain amount of pain in the affected limbs may still be present; in some cases, especially the milder ones, pain persists for days and even weeks. By this time the amount of paralysis has become evident, but this is generally reduced in the course of the next two or three weeks, and those muscles which have been but little affected are rapidly restored to activity. There remain a number of muscles, varying in every case, which are paralysed for a period of many months, if not permanently. These soon show evidence of atrophy and, on electrical examination, present the reaction of degeneration. All tendon-jerks in which the paralysed muscles are concerned disappear, and the presence or absence of superficial reflexes depends on the state of the muscles involved in them. If plantar responses are obtained they may be either flexor or extensor in type. Cutaneous sensibility is rarely if ever affected, but muscles which are moderately paralysed are usually tender on manipulation. If there has been any difficulty with micturition or defaecation in the acute stage of the disease it has now disappeared and the sphincters have regained their normal functions. Lumbar puncture during the first week of the patient's illness generally finds the spinal fluid at high tension and often containing a small excess of lymphocytes.

In more virulent examples of the disease all four limbs and the trunk muscles become rapidly and completely paralysed, with the result that the child depends on its diaphragm

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and accessory muscles for respiration. If this distressing condition is not rapidly ameliorated, an attack of bronchitis is likely to bring about a fatal termination. In other instances the paralysis is even more widespread and, by involvement of all respiratory muscles, and perhaps of those innervated by the bulb, kills the patient in the course of thirty-six to forty-eight hours. As some meningeal symptoms may be present the diagnosis of poliomyelitis may not be considered in such instances, and the whole condition may be attributed to meningitis.

Stage of repair.—During the stage of repair, which may be said to last from the end of the second or third week after the acute attack for a year or eighteen months, the amount of permanent muscular atrophy can be determined. While some muscles remain shrunken and useless, others, after a period of paralysis and atrophy, become slowly restored to a condition which may approach the normal. The effects of the muscular atrophy on the growth and development of the limbs also become apparent. The skin covering the peripheral parts of paralysed limbs tends to become cold, purple and either unusually dry or persistently moist. Unless the utmost care is taken with the limbs, alterations in joints and contractures of muscles combine to produce serious deformities. Generally speaking, the bones become relatively shortened and somewhat brittle, although in rare instances traction on the epiphysis may lead to the elongation of one or more of the long bones. At least a year must pass by before one can say with certainty which muscles are likely to recover. Those which retain some faradic excitability may be depended upon to recover more or less completely, while those which present the complete reaction of degeneration may after many months show a quickened response to the galvanic current and some return of faradic excitability, giving rise to the hope that a degree of functional activity may be restored.

No statement of any importance can be made as to the incidence of the paralysis in various regions of the body, but the legs are more often affected than the arms, and the limbs than the trunk. The muscles which dorsiflex the ankle, the extensors of the knee and the extensors of the hip are more often affected than their antagonists, while in the upper limb the muscles concerned with movements at the shoulder and elbow suffer more

often than the intrinsic muscles of the forearm and hand. Asymmetry is the rule, and may almost be considered a characteristic feature of the disease.

Permanent results.—At the end of eighteen months or two years the patient who has received adequate care and treatment will have regained all the active muscular tissue which is possible, and his disability can only be modified by orthopedic measures. On the other hand, cases which have been neglected during the first year or two may later respond to more efficient treatment.

Diagnosis.—In young patients a febrile illness associated with painful and more or less inactive limbs may be the result of rickets, infantile scurvy, syphilitic epiphysitis, osteomyelitis, or rheumatic fever, and in some cases the diagnosis from acute poliomyelitis may be difficult. An accurate conclusion can only be arrived at by detailed examination, including that of the reflexes. Symptoms suggesting *meningitis*, such as rigidity of the spinal muscles and the presence of strabismus, may be misleading, but examination of the cerebro-spinal fluid will put an end to any doubt. The more gradual onset, the symmetrical distribution of the palsy in peripheral parts, and the presence of some sensory disturbance usually suffices to distinguish *multiple peripheral neuritis* from poliomyelitis, in spite of the fact that the former condition may sometimes fail to show any great disorder of sensibility. In adults the rare condition known as *Landry's paralysis*, with its gradual slow involvement of the musculature and little constitutional disturbance, has been mistaken for acute poliomyelitis. The diagnosis between these two conditions is not always easy.

Prognosis.—The mortality of poliomyelitis in the acute stage is not high, and in any particular instance the prospects of recovery must depend upon whether the respiratory muscles are seriously affected. In other words, a fatality is the result, either of complete respiratory failure or of an acute pneumonic or bronchial infection, in a patient whose respiratory musculature has been partially damaged. The prognosis in regard to the recovery of muscular power cannot be determined with certainty until some months have elapsed, and then depends upon the presence or absence of atrophy and the response of the affected muscles to electrical stimulation, the consideration being given to the treatment which has been employed.

POLIOMYELITIS, ACUTE

Treatment.—In the presence of an epidemic, avoidance of contact with patients suffering from the disease is the only prophylactic measure which can be recommended, and in view of the probability that healthy persons carry the infection, it cannot be regarded as a very adequate method of protection. If the possibility of poliomyelitis is suspected when a child develops a febrile disturbance, it may be worth while administering hexamine by the mouth. The treatment of the acute stage is similar to that of other acute specific fevers. Severe pain may be relieved by salicylates, phenacetin, antipyrin, or even by an injection of morphia. If retention of urine is present, catheterization may be necessary during the first few days. In the presence of respiratory embarrassment inhalations of oxygen are indicated, and it may be necessary to carry out artificial respiration.

As soon as the general condition of the patient permits, gentle passive movements of the paralysed parts should be performed several times in the twenty-four hours, and changes in the position of the limbs should be frequently made in order to avoid the development of contractures. Every method should be employed to encourage the patient to attempt voluntary movement of the affected muscles, and in the case of infants a certain amount of ingenuity must be exercised in order to attain this end. Paralysed parts must be kept warm with suitable woollen garments, and the circulation stimulated by massage at frequent intervals.

The use of electrical current is of importance, but it must be remembered that no electrical treatment is of any value unless it stimulates muscles to contract. The galvanic current should be employed, the kathodic electrode being applied to the paralysed muscles. Careful attention must be given to the position of the limbs, having regard to the principle that a paralysed muscle will not regain its activity if it is kept on the stretch. Various artificial instruments are used for this purpose; the continuous abduction of the arm by some form of splint in order to relax a paralysed deltoid may be cited as an instance. In children with the lower limbs affected it is important to postpone active locomotion until the muscles of the legs are sufficiently strong to prevent the development of abnormal positions and deformities. Light splints made of celluloid have been largely employed with the idea of allowing active movements and of preventing the adop-

POLYORRHOMENITIS

tion of unnatural postures. No general rule can be laid down with regard to orthopædic measures, since every case must be carefully considered on its own merits.

E. FARQUHAR BUZZARD.

POLIOMYELITIS, CHRONIC (see MUSCULAR ATROPHY, PROGRESSIVE).

POLITZERIZATION (see EAR, EXAMINATION OF).

POLYCYSTIC DISEASE (see KIDNEY, CYSTS OF).

POLYGRAPH (see HEART-BEAT, ABNORMALITIES OF).

POLYMYOSITIS HÆMORRHAGICA (see MYOSITIS).

POLYNEURITIS (see MULTIPLE NEURITIS).

POLYORRHOMENITIS (*syn.* Polyserositis, Multiple Serositis).—A multiple inflammation of the serous membranes, subacute or chronic in character, affecting the peritoneum, both pleuræ, and the pericardium, either simultaneously or, more often, successively. As affecting more particularly the pericardium, the syndrome is described under PERICARDITIS.

Etiology.—The affection occurs both in childhood and in adult life, the sexes being affected about equally. Its exact nature is obscure and some cases are quite unexplained, but it is probably an attenuated infective process which may be set up by various micro-organisms or their toxins. In a good many instances it is tuberculous in nature and associated with definite caseous changes, the disease starting perhaps from the mediastinal glands or, in women, from the Fallopian tubes, but never involving the viscera. Sometimes it is rheumatic in origin and then usually begins by affecting the pericardium. Other pathogenic microbes—pneumococci, streptococci, etc.—may at times be the causal agents. Although it may last for several years, its tendency is slowly to progress, sometimes with subacute exacerbations.

Pathology.—The affection leads to much fibrosis and contraction, and especially to thickening of the mediastinal tissues. The heart may be dilated, or smaller than usual, owing to the strangling action of the newly formed fibrous tissues which envelop it. Both pleuræ are often affected, especially the right, and a sero-fibrinous effusion may form from time to

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time, but eventually the two pleural layers become firmly adherent and greatly thickened. Owing to interference with the movements of the diaphragm the bases of the lungs tend to become collapsed. In the peritoneum, where the disease often begins, the most pronounced feature is perihepatitis, which gives rise to ascites. The hepatic veins may be narrowed and the liver is often enlarged, hard, and shows fibrous and congestive changes. The spleen also is often enlarged and shows perisplenitis.

Symptomatology.—Often the first obvious indication of disease in these patients is ascites, but sometimes the signs of adherent pericardium are pronounced, especially upward extension of dullness. Later, they present the ordinary features of gradual heart failure, with cyanosis, distension of superficial veins, increasing dyspnoea and orthopnoea, and congestion of the pulmonary bases. Albumin is often present in the urine, and perhaps chronic interstitial nephritis is a predisposing factor in some of the cases in which the disease begins in the peritoneum.

Treatment can only be symptomatic. Ascites often requires repeated tapping, and less frequently a pleural effusion calls for paracentesis. Heart tonics, especially digitalis, are valuable for maintaining the vigour of the circulation. In tuberculous cases death is often due to tuberculous meningitis.

J. WALTER CARR.

POLYPUS (see NASAL POLYPUS; UTERUS, POLYPI OF; RECTUM, NEW GROWTHS OF).

POLYSEROSITIS (see POLYORRHOMENTIS).

POLYURIA (see URINE, VARIATIONS IN AMOUNT OF).

POMPHOLYX (*syn.* Cheiopompholyx; Dysidrosis).—A vesicular eruption of the hands and feet which has a tendency to seasonal recurrence.

Etiology.—This disease is sometimes regarded as a form of eczema, but its clinical appearance, course and pathology justify its classification as a distinct affection. The attack is usually associated with some occupation which leads to excessive sweating of the parts involved. Patients attacked with this complaint are often of nervous disposition or in indifferent general health.

Pathology.—Those who regard the disease as an eczema maintain that the lesions are inflammatory. An older view, that the vesicles

are primarily non-inflammatory and due to an accumulation of sweat in the epidermis, has received support from some observations of Nestorowsky, who from examination of 2,800 microscopical sections in six cases concluded that the vesicles are due to collections of sweat dammed back by swelling of the horny cells which border the mouths of the ducts. The swelling of the horny cells he regards as a result of the action of a toxic sweat.

Symptomatology.—The outbreak of the eruption is often coincident with the sudden onset of hot weather. It is accompanied by burning, tingling and itching. The hands are usually more severely attacked than the feet, but occasionally the feet may suffer more, or the hands or the feet may be alone attacked. The eruption appears as deeply-seated vesicles along the interdigital spaces and in the palms, or on the sides and soles of the feet, and these earlier or smaller vesicles are well described as "like boiled sago-grains embedded in the skin." These slight eruptions are often called *dysidrosis*, and the term pompholyx, or cheiopompholyx, is generally used for more severe and extensive cases in which the vesicles enlarge to bullae, and sometimes involve the whole hand or foot on palmar or plantar and dorsal surfaces. In such cases the vesicles may be so closely massed that they coalesce to form loculated bullae. The vesicles and bullae remain for several days without rupturing and eventually dry up, leaving the roofs of the bullae as brownish scales, which in course of time separate and display the newly-formed red and tender epidermis.

The bullae may, however, become secondarily infected, the contents turbid, and the skin at their base inflamed; from this infection a secondary impetigo may spread to the arms and other parts. On subsidence of the eruption the patient may be free until the next summer, or for several years, or there may be no subsequent attack, though a repetition is usual.

Diagnosis.—The affections likely to be confused with dysidrosis or pompholyx are scabies, eczema and eczematoid ringworm. The limitation to the hands and feet and the absence of the burrows of the acarus exclude scabies. In eczema the vesicles are smaller, and set upon a red swollen area; itching is a prominent feature, and the eruption does not dry up spontaneously, but may continue for months or years unless appropriately treated. Eczematoid

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ringworm of the hands and feet may closely simulate pompholyx. If the vesicular eruption is localized or patchy, with sharply-defined borders, ringworm should be suspected, and a search made for fungus in the scales by their examination in liquor potassæ under the microscope.

Treatment.—The affected parts should be soaked for half an hour, several times daily, in a bath of hot boric-acid lotion. After the bath, lotio calaminæ should be applied, or in severe cases a zinc ointment thickly spread on butter muslin and lightly bound on. The general health must be attended to. Lotio calaminæ may be used in hot weather to prevent fresh attacks. A general tonic is often of great service.

H. G. ADAMSON.

PONTINE HÆMORRHAGE (*see* NERVOUS SYSTEM, CENTRAL, LOCAL LESIONS OF).

PONTINE TUMOURS (*see* CEREBRAL TUMOUR).

POSTANÆSTHETIC VOMITING (*see* VOMITING, POSTANÆSTHETIC).

POSTERIOR BASIC MENINGITIS (*see* MENINGITIS).

POST-HEMIPLEGIC CHOREA (*see* ATHE- TOSIS AND TREMOR).

POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES.—In non-judicial cases (e.g. bodies of persons who have died in hospital, etc.), and in cases likely to involve civil litigation, no further authority for making a necropsy is required than the consent of the nearest relative or guardian; it is always advisable to obtain this in writing. In criminal cases the authority is the summons of the Coroner (in England) or the warrant of the Sheriff (in Scotland). When there are any circumstances which necessitate inquiry by the Coroner or Procurator-Fiscal, a medical man must not make a post-mortem examination without the necessary written authority. If evidence of unnatural death is unexpectedly found during the course of a necropsy undertaken for purely professional reasons, the examiner must notify the authorities. In such circumstances no fee will be recoverable, as the examination was not undertaken at the request of the Coroner or Procurator-Fiscal.

Before the examination is begun, the body should be identified by two or more competent witnesses. Whenever possible there should be

personal identification, but where this cannot be done there must still be identification of the body as that of an "unknown person found in a particular place at a specified time." In either case the names, designations and addresses of the persons identifying the body should be noted.

Notes of everything found should be written down on the spot, and if two medical men are making the examination it is convenient for one to do the dissection and for the other to take notes. As their report will be based on these notes, both examiners should revise them when the dissection has been completed (*see* MEDICAL EVIDENCE AND REPORTS ON MEDICO-LEGAL CASES).

In every case the examination should be complete, since it may easily be suggested by counsel that a natural cause of death existed in an organ that was not examined. Unless a complete examination has been made the witness will be unable to disprove the counsel's suggestion.

External examination.—This should have reference to—

(a) Points which will assist in establishing identity in those cases in which the body has not been identified in the personal sense (*see* below).

(b) The clothing. Note should be made of the number, position, and size of stains of mud, blood, or other material. Any cuts in the clothing should be examined to determine if they correspond to wounds present on the body. In the case of new-born children note should be taken of any clothing or wrappings present on the body. If a special examination is made of stains on clothing, a separate report should be given.

(c) Appearances indicating the time of death and the position in which the body has been lying after death, e.g. rigor mortis, hypostasis, and putrefactive changes (*see* below).

(d) Marks of violence and other signs indicating the cause of death. The most important of these are wounds and bruises, marks of ligature on the neck, burns and scalds, scorching of skin and marks of powder particles round gunshot wounds (not always present), foreign bodies in air-passages, fractures and dislocations, ligaturing of hands and feet, the presence of objects grasped in the hand and of mud, etc., beneath the finger-nails. Wounds should be accurately measured, their characters noted, and their situation described with reference to definite anatomical landmarks. The description of

POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES

bruises, burns, etc., should be correspondingly complete. (See LIGHTNING AND ELECTRIC DISCHARGES, INJURIES AND DEATHS FROM.) The vagina and rectum as well as the air-passages should be examined for foreign bodies.

Internal examination.—The cranial cavity and the cavities of the thorax and abdomen are opened by the customary incisions. Should a wound be present in the line of the usual incision, the incision should be deviated so that after opening the body the wound can be examined in its whole course. In opening the skull the saw only should be used, as the chisel might fracture the bone or lead to the extension of an existing fracture.

The attention given to a particular organ or region will depend on the conditions found, but a systematic examination of all the organs is necessary. Some authorities consider that a better idea is obtained of the condition of the intracranial venous channels if the cranial cavity is examined before the organs of the thorax and abdomen are removed and the cranial vessels drained by division of the large veins. There is probably very little in the claim, but the method should be adopted, as it will prevent the point from being raised in cross-examination. After the brain has been removed and examined by sectioning, the dura should be stripped from the bone and the skull examined for fractures. At this stage the finger may be slipped into the foramen magnum in search of any dislocation of the upper cervical vertebrae.

The organs of the thorax and abdomen should first be examined *in situ*, and then removed and examined individually. Any blood, pus or other fluid present in the cavities should be noted, and the amount measured. The examination of the organs is carried out in the usual post-mortem room fashion. In medico-legal examination, however, special attention should be given to certain points. The amount and character of the stomach contents should always be noted, and also the amount of urine in the bladder. It is well, too, to test the urine for the presence of sugar, etc. The stomach, duodenum, etc., may be coloured by bile which readily permeates the tissues, or may be discoloured from hypostasis, and care should be taken to differentiate such markings from signs of inflammation or putrefaction. The inner surface of the ribs should always be examined for fractures, and the vertebral column for fractures and dislocations. The trachea, larynx, oesophagus, rectum, and vagina

should be laid open and examined. Great care must be taken in probing the track of a wound; and no more force should be used than is required to allow the probe to slip along the track.

In cases of suspected poisoning, a special procedure must be followed. Careful attention should be given to the whole of the alimentary tract from the mouth downwards for evidence of staining, erosion, or inflammation, and any unusual odour (chloroform, hydrocyanic acid, etc.) detected on opening the abdomen should be noted. The stomach and intestines are separated between double ligatures and removed. The stomach is then placed in a perfectly clean flat dish, opened, and examined. The small and large intestine is treated similarly. The organs and their contents are then placed in perfectly clean glass jars, having tight-fitting covers and without any preservative being added, the stomach in the first, the stomach contents in the second, and so on. In other jars are placed the liver (or a portion of the organ), kidneys, spleen, brain, and other organs removed, and also the urine and a sample of blood. The jars are then covered, sealed, labelled so that they can be identified, and stored in a lockfast place till they are handed over to the analyst. A note should be kept of the number of jars and their contents, and a receipt obtained for them when they are handed over. (For further details, see Post-mortem Evidence, under POISONS AND POISONING, p. 593.) It should be remembered that, except for corrosives and irritants, poisons may destroy life without leaving any naked-eye changes.

Identification of the dead.—If the body has not been identified in the personal sense, the points which require the attention of the examiners are the sex, height, hair, apparent age, congenital deformities and birth-marks, and injuries leaving permanent results.

Sex.—Difficulty will only arise (a) in cases of hermaphroditism and pseudo-hermaphroditism (see HERMAPHRODITISM), (b) when the body is much decomposed, and (c) when mutilated remains without sexual organs are being examined. Generally, the male is of stronger build than the female, and the adult male is broader at the shoulders than at the hips, the adult female the reverse. But general build is not always a safe means of distinguishing the sexes, since there are masculine females and effeminate males. In females the pubic hair is usually confined to the mons veneris, but in males it frequently extends on to the abdomen.

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in the latter, hair may be present also on the chest. In decomposed bodies the sexual organs should always be looked for, and it should be remembered that the uterus is one of the last organs to putrefy. Male sex has been established from a portion of corpus cavernosum penis still attached to the ischium. If no sexual organs are found, attention should be given to the distribution of hair on the body, the character of the mammary glands, and the presence or absence of lineæ albicantes from previous pregnancy. Help may be derived from the bones. The bones of the female are generally lighter than those of the male; but the greatest assistance is obtainable when the pelvis is available for examination. As compared with the male pelvis, the female pelvis is shallower, the cavity more capacious, the bones smoother and less massive, the iliac crests spread farther apart, the sacrum is wider and flatter and the sacral promontory less prominent, the arch of the pubes is wider, and the transverse, oblique and antero-posterior diameters of the pelvis are greater. In addition, the angle which the neck of the femur makes with the shaft is obtuse in the male and more nearly a right angle in the female.

Height.—An accurate measurement of height can always be made when examining a complete body. When dealing with bones, a fair estimate may be made if the vertebral column, skull, and one leg are available. The bones are placed in their correct anatomical position, the height measured, and one and a half inches added in respect of the soft parts.

Hair.—Colour, length and distribution should be noted, and also whether the hair has been dyed. Dyed hair loses its lustre, the colour is often not uniform, and the scalp frequently shows the effect of the dye. The colour of the hair on the head should be compared with that of the hair on other parts of the body.

Age.—The estimation of age is not always easy in fresh bodies, but becomes more difficult as decomposition advances. In the most favourable circumstances, only an approximate estimate can be given. In the young, general development, height, weight, eruption of teeth, centres of ossification, and the union of epiphysis in the long bones will assist. It is in middle life that the greatest difficulty is experienced. The presence of the wisdom teeth and union of the epiphysis to the shaft in all the bones is evidence that adult life has

been reached. At later life an estimate (subject to considerable error) may be made by considering—

(a) The angle between the neck of the femur and the shaft—obtuse in middle life and nearer a right angle in old age.

(b) The angle between the horizontal and vertical rami of the lower jaw—nearly a right angle in middle life when the molar teeth are present, and more obtuse in the edentate jaw of the aged.

(c) The teeth—generally lost with advancing age, but may have been extracted. The number and condition of the teeth is of importance in ordinary personal identification.

(d) Degenerative changes—calcareous deposits in the costal, tracheal, and laryngeal cartilages, atheromatous changes in the arteries, thinning of the bones of the skull from absorption, and the presence of the arcus senilis.

Congenital deformities and birth-marks.—The importance of nævi, cleft palate, hare-lip, supernumerary digits, supplementary mammæ, etc., in personal identification is apparent and need not be discussed.

Injuries leaving permanent results.—Scars, tattoo marks, ununited or badly united fractures, amputations, operations on joints, etc., all fall under this heading.

Scars are composed of fibrous tissue covered on the surface by a few layers of epithelium. Where there has been no breach of continuity of the true skin, e.g. superficial scratches, the scar may disappear, but where the true skin has been involved the scar is *permanent* and can only be removed by excision or other method, with the production of a new scar.

Tattoo marks may or may not be permanent. They are made by puncturing the skin with needles dipped in colouring matter, the pigment being deposited in the deepest layer of the epidermis or the superficial layer of the true skin. Disappearance is more likely when the pigment is confined to the former situation. The black or blue colours are generally produced by gunpowder, indian ink, charcoal, common ink, prussian blue, and the red colours by vermilion or cochineal. The black colours are more permanent than the red, and marks produced by carbon in the true skin probably never disappear naturally. If a tattoo mark is suspected to have disappeared, the neighbouring lymphatic glands should be examined for pigment. The artificial removal of marks will produce destruction of the true skin and lead to the formation of a scar.

POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES

Signs indicating the time of death, and the position in which the body has lain after death.—The most important are cooling of the body, rigor mortis, hypostases, contact flattening of muscles, and putrefaction.

Cooling of the body.—After death there is a progressive fall of bodily temperature till equilibrium is reached between the temperature of the body and that of the surrounding medium, the rate being proportional to the difference between the two. In the air, the body usually becomes cooled to the temperature of its surroundings in 12–24 hours. The initial is more rapid than the subsequent loss, and the average rate for each of the first twelve hours is 1° C. Cooling is delayed by acute pyrexial diseases, clothing, obesity, asphyxia, sudden death in health, and high temperature of the air. In water, cooling is more rapid than in air. In deaths from cholera and acute rheumatism the temperature has been observed to rise, owing probably to microbic action.

Rigor mortis.—Death-stiffening, which sets in some hours after death, is almost invariably present, but may be overlooked in a particular case from being small in amount or transient. It is due to the formation of myosin or muscle-clot from the muscle proteins, myosinogen and paramyosinogen. It appears more rapidly in the involuntary muscles, and the ventricles may be found hard within an hour of death. The usual order and average time of invasion of the voluntary muscles is—neck, jaw, and face, 5–6 hours after death, definitely present in the upper part of the body in 10 hours, present all over the body in 12–18 hours; it has generally disappeared in 36 hours. After death from enteric fever it has been reported present 3½ minutes after death; in that case it disappeared in 15 minutes, and decomposition set in within an hour of death. On the other hand, it has been known not to appear for 16 hours, and has been found present 21 days after death. Parts of the body lose the stiffening in the same order as they were affected. As a rule, the earlier rigor mortis comes on the sooner does it pass off, and the later its appearance the longer will it persist. The onset is hastened when the muscles have been in an exhausted condition before death, e.g. after great exertion, and in deaths from infectious and wasting diseases. In cases of poisoning by strychnine and other convulsant poisons the onset may be very rapid and (contrary to the usual rule) the duration long. The condition persists longer during

cold weather than in a warm moist atmosphere. If a body already the subject of rigidity is exposed to a temperature of 75° C., albuminates other than myosin are coagulated and a higher degree of rigidity, known as “heat-stiffening,” results. It is sometimes necessary to distinguish rigor mortis from the stiffness due to freezing. In the latter condition all the tissues, including the skin, are hard and rigid, and the joints give a crackling sound when forcibly flexed, whilst in death-stiffening the skin remains soft and unaffected.

The terms “cadaveric spasm” and “instantaneous rigor” are employed to designate the instantaneous stiffening of the body or part of the body in the position in which it was at the moment of death. When this occurs it allows definite conclusions to be drawn. Thus, a knife or other weapon found firmly grasped in the hand of a person with a fatal wound is conclusive evidence that the weapon was so grasped at the moment of death.

Contact flattening.—This takes place in those parts of the body which lie in contact with any hard surface, and therefore gives an indication of the position the body has lain in after death.

Hypostases or cadaveric lividity.—The discoloration of the tissues to which these names are given is due to the blood, while still fluid, gravitating into the capillaries and small vessels of the lower part of the body. Its situation on the body will therefore indicate the position in which the body has lain for some time after death. Hypostases are of constant occurrence, and may be found even after death from hæmorrhage. They are found in the internal organs as well as in the skin, and should not be confused in the former situation with signs of inflammation or other pathological changes. They are not found on that portion of the skin which bears the weight of the body, the pressure preventing the blood from entering the capillaries. The staining is usually well marked in 8–12 hours after death, but may be observed as early as the fifth hour. There is at first a patchy distribution of lividity, giving a mottled appearance, but later on the smaller areas coalesce. The patches do not project above the surface of the skin, and should be distinguished from bruises. If a bruise is incised, extravasated blood will be found in the tissues, whilst incision of an hypostasis will show no such extravasation and only slight oozing from the cut mouths of the vessels. Never confound them

POST-MORTEM EXAMINATIONS IN MEDICO-LEGAL CASES

you have found a bruise unless you have verified it by incision.

Putrefaction.—The decomposition of dead bodies is due to the agency of micro-organisms, all of which require the presence of moisture and a suitable temperature. To some, oxygen is necessary (aerobic), others can do without oxygen (anaerobic). The disappearance of the body may also be assisted by flies, rats, fish, etc. The condition of a body some time after death will therefore depend on a wide variety of circumstances. As decomposition proceeds, gases of putrefaction are evolved and distend the tissues and the body cavities. Finally, the tissues and the cavities of the body burst open, the soft parts are changed into a pulsatous green mass, and the bones are exposed. This is the stage of *colliquative putrefaction*, and is usually reached in this country in from two to five months. In bodies exposed to air the earliest signs of decomposition observed externally are a brownish coloration of the surface veins and a greenish or bluish coloration of the skin, usually first observed in the right iliac region of the abdomen. Later, the eyeballs soften and then liquefy, the greenish coloration extends over the body, bullæ appear on the surface, the abdomen becomes distended with gas, maggots appear in the orbits, on the scrotum and other parts, the nails loosen and fall off, the face becomes swollen, bloated, and unrecognizable. In a body which has lain in water there is first bleaching of the skin of the palms and soles, then swelling and discoloration of the skin over the face, neck, and sternum. Later, the swelling and discoloration extend to the shoulders, arms, abdomen, and legs.

Since the process of putrefaction is influenced by so many circumstances, the date of death can be estimated only approximately from the stage of decomposition. Some of the internal organs putrefy early, others late, and this gives some assistance. According to Casper, the order is as follows:

Organs which putrefy early—

Larynx and trachea, bright red or greenish in 3-5 days.

Brains of infants, soft and pulpy in 4-5 days or less.

Stomach and intestines show signs in 5 or 6 days.

Spleen soon becomes soft and dark.

Liver soon superficially discoloured, but firm for some weeks.

Brain of adults firm for a week or two.

Organs which putrefy later—

Heart, distinguishable for months.

Lungs, gas bubbles between pleura and lungs often seen early, but no further decomposition even for months.

Kidneys and bladder, œsophagus, pancreas, diaphragm, blood-vessels, and uterus, approximately in the order stated.

The chief circumstances influencing the onset and progress of putrefaction are:—

(a) *Internal*: Age, sex, condition of body, and kind of death.

Children decompose more readily than adults. Though putrefaction proceeds rapidly in the bodies of women who have died from puerperal sepsis, sex *per se* has no influence. Fat flabby bodies putrefy more quickly than those which are thin and emaciated. Putrefaction is rapid after death from asphyxia, and when death has been due to acute disease, particularly septic disease. Parts which have been mutilated or inflamed decompose rapidly. Putrefaction is inhibited when death has been due to poisoning by arsenic, antimony, carbolic acid, or chloride of zinc. In the case of arsenic and antimony the body may be preserved for several years.

(b) *External*: Access of air, temperature, medium in which body is lying, moisture.

Putrefaction is promoted by the presence of air and retarded by its absence. Other things being equal, decomposition will be quicker in a nude body than in one which is clothed, in a body buried in sandy soil than in one buried in clayey ground, in a body buried in a flimsy coffin than in one sealed in a lead shell. Putrefaction goes on most rapidly between 70° and 100° F., but will commence at any temperature above 50° F. The temperature of boiling water sufficiently long maintained will sterilize animal tissues, whereas a temperature near the freezing point arrests bacterial action but does not sterilize. According to Casper, a body decomposes in air twice as quickly as in water, and eight times as quickly as in the earth. Moisture is essential for microbic action, and the animal body possesses sufficient water for the process of putrefaction.

Saponification.—This change may occur in bodies which have been exposed to moisture, either from long submersion in water or burial in damp soil, and has occasionally been seen even in a body shut up in a trunk. The fat and other soft tissues are changed into a soft, yellowish-white, waxy-looking substance called *adipocere*, which is a soap having ammonia,

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potassium, sodium, and calcium in varying proportions as the base, and oleic, stearic, butyric, or other fatty acid as the acid. It is not usual for the whole body to undergo the change; the breasts and buttocks appear to be specially susceptible. After submersion in water, *traces* of adipocere have been found in four or five weeks, but eight to twelve months are required for its development in damp soil.

Mummification.—For this change a free circulation of warm dry air is necessary to remove the moisture present in the tissues. Putrefaction is thereby prevented, and the body, though shrivelled and black, preserves its anatomical characters. The condition occurs readily in warm countries with a dry sandy soil, and in this country has been met with in the bodies of infants concealed in places which allowed the necessary factors to come into play, e.g. behind kitchen grates.

A. ALLISON.

POST-MORTEM WART (see SKIN, TUBERCULOSIS OF).

POST-PARTUM HÆMORRHAGE (see HÆMORRHAGE, POST-PARTUM).

POSTOPERATIVE SHOCK (see SHOCK AND COLLAPSE).

POTASSIUM BICHROMATE, POISONING BY (see POISONS AND POISONING).

POTASSIUM CYANIDE, POISONING BY (see POISONS AND POISONING).

POTT'S DISEASE (see SPINAL CARIES).

PRECIPITIN TESTS (see SEROLOGICAL DIAGNOSIS; BLOOD-STAINS, EXAMINATION OF).

PREGNANCOY, EXTRA-UTERINE.—Extra-uterine or ectopic gestation signifies the fertilization and embedding of an ovum in some site other than the uterus. Usually this site is the Fallopian tube, but a certain number of cases have been described in which the ovum has been fertilized and has developed in the ovary itself—ovarian gestation. Primary abdominal gestation in which the ovum has developed from the first in the peritoneal cavity has been described, but no case has as yet been completely proved. As will be shown later, the peritoneal cavity is often the site of a secondary gestation sac after rupture of the tube or escape of the ovum through the abdominal ostium.

Etiology.—The causation of extra-uterine

pregnancy is not fully understood, but in most cases there can be no doubt that it is an accidental circumstance. The ovum normally is fertilized in the tube and begins to develop there. By the time it passes through the narrow isthmic portion into the uterus it must have attained such a degree of development that it is covered with trophoblast and is able at once to embed itself in the uterine mucosa. It is not difficult to imagine that sometimes the ovum is so large that it cannot pass through the narrower portions of the tube. The older view that the condition is the result of inflammatory changes in the tube—salpingitis—is untenable, as numbers of cases occur in which no such changes can be discovered. Kinks in the tube due to adhesions have been suggested as causes, but are unlikely ones, because adherent tubes are nearly always closed and consequently could not receive an ovum. Occasional diverticula of the tube occur, and sometimes harbour a fertilized ovum, but they must be very rare.

Anatomy of a pregnant Fallopian tube.

—The fertilized ovum does not remain in the lumen of the tube, but bores its way through the epithelial lining into the very scanty connective-tissue layer beneath. The ovum thus becomes embedded in the wall of the tube, just as under normal conditions it embeds itself in the uterine wall. By the time that the ovum is embedded, the opening by which it entered the tube-wall is smaller than itself, for its development has been continuing. Thus there must be a portion of the tubal mucous membrane raised up over the ovum, forming what is known as the capsularis. As the ovum bores its way into the muscle-coats of the tube, this capsularis may even consist partly of unstripped muscle. The connective tissue beneath the epithelium is very small in amount, and fails to undergo a complete decidual reaction, keeping pace with the growth of the ovum as the uterine connective tissue does. The ovum must therefore bore its way progressively into the muscle-coats until the peritoneum is reached. This, the result of the eroding action of the trophoblast, constitutes the great danger of tubal gestation, for, together with the distension of the tube, it must eventually lead to rupture when the coats become sufficiently thinned.

Morbid results of tubal gestation.

Both the ovum and the tube are affected. The first untoward result is *hæmorrhage* around the ovum itself, into the wall of the tube, and also

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into its lumen. This hæmorrhage is the result of the erosion of blood-vessels by the action of the trophoblast, and its usual effect is to convert the ovum into a tubal mole, by welding all the chorionic villi into a solid mass by laminated bloodclot. The amniotic sac remains intact and does not rupture, however great the hæmorrhage; it can generally be recognized in the mole. Although the formation of a mole is common, it is not invariable, especially in cases of rupture of the tube. Hæmorrhage into the wall of the tube ploughs up the muscle-coats, and helps in its local destruction, while bleeding into the lumen increases the tension and therefore facilitates rupture.

The final morbid effects are *tubal rupture* and *tubal abortion*; the latter is much more common than the former, but is not nearly so serious or so likely to lead to fatal hæmorrhage. The mechanism of *tubal rupture* has already been described; it is a combination of erosion of its wall with increased tension within its lumen, the result of the growth of the ovum and of hæmorrhage. Tubal rupture gives rise to free hæmorrhage because the sudden tearing of the wall of the tube opens an artery of considerable size whose walls may be so damaged as to be incapable of retraction and closure. In *tubal abortion* the ovum is extruded, nearly always in the form of a mole, through the abdominal ostium into the peritoneal cavity. This can only happen when the ovum is developing in the wall of the tube somewhere near the fimbriated extremity. As the ovum enlarges in such a case, the tube-walls are expanded, and the abdominal ostium is gradually drawn towards the ovum and stretched open. When at length the ostium reaches the widest diameter of the ovum, the latter mechanically slips, or is squeezed out by contraction of the muscle-coats. The incident is always accompanied by hæmorrhage, the blood accumulating rather slowly and clotting in layers around the ovum and the fimbriated end of the tube, so that the two are often welded together and form what is known as a peritubal hæmatocele. By far the larger number of tubal gestations end in this way, a peritubal hæmatocele being the usual condition found at operations. When hæmorrhage is severe, as in rupture of the tube, the blood is poured out into the abdominal cavity and does not clot at once; its amount may be so great as to fill the abdomen and cause death before clotting has begun. Should, however, the hæmorrhage cease sponta-

neously, the blood in the upper part of the abdomen is rapidly absorbed, while that in the pelvis clots and forms a pelvic or a retro-uterine hæmatocele. A pelvic hæmatocele varies in size, and often reaches as high as the umbilicus; it is accompanied by inflammatory changes in the peritoneum and consequently by adhesions which fix all adjacent structures to it. By this means the omentum and the small intestine always become adherent to a hæmatocele.

Further development of the ovum after tubal abortion or rupture.—As a rule, the ovum dies when it escapes from the tube, but occasionally the escape is not complete, some part of the chorionic sac retaining its attachment to the tube-wall. This may suffice to keep the ovum alive whilst it is forming new attachments outside the tube. It is essential in such a case that the amniotic sac should be intact; if this is not so, death of the embryo always occurs. When the ovum continues to live, a *secondary abdominal* or *secondary intraligamentary gestation* results; abdominal when the ovum escapes into the peritoneal cavity either through the abdominal ostium or through a rupture in the wall of the tube; intraligamentary when the tube happens to rupture between the layers of the mesosalpinx. In either situation the ovum may continue to develop until the fœtus has reached full-time, or secondary hæmorrhage may occur from accidental detachment of the placenta, with great risks to the patient and death of the fœtus. In secondary abdominal gestation peritoneal adhesions form around the fœtal sac and fix all adjacent structures, while the placenta continues to develop; it is usually situated at the bottom of the pelvis and attached to the tube, broad ligament, lateral pelvic wall, rectum, etc. Secondary intraligamentary gestation is interesting because the gradual growth of the ovum opens up the layers of the broad ligament, lifts up the peritoneum from the pelvis, and may eventually strip it from the abdominal wall to a variable height. Sometimes it is possible to open the fœtal sac through the abdominal wall without opening the peritoneal cavity.

When at any period of its development a fœtus dies in a secondary gestation sac, it may become converted into a stony mass known as a *lithopædion*. It is produced by deposition of lime salts in the fœtal membranes and on the surface of the fœtus. More rarely the whole fœtus is calcified and forms a homogeneous

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mass. On the other hand, the sac may become infected and suppurate, the consequent abscess bursting into some viscus such as the bladder or intestine. In these circumstances the foetus may disintegrate, its bones being gradually discharged while the patient suffers from severe and usually fatal toxæmia. Death may also be due to lardaceous disease, the result of prolonged suppuration. Now that extra-uterine gestation is usually recognized and dealt with by operative means, such results have become very rare.

Clinical results and symptoms.—*Rupture* or *abortion* usually occurs at an early date. The duration of a tubal pregnancy rarely exceeds six weeks, and is often much less; consequently the symptoms of pregnancy are seldom very pronounced. The patient may have missed one menstrual period, and a week or two later is suddenly seized with acute abdominal pain, faintness, vomiting and collapse. In rare cases death occurs within a few hours, but more usually the patient gradually recovers, and hæmorrhage from the uterus begins and continues for a variable period, not uncommonly for several weeks. A decidua always forms in the uterus in sympathy with the developing embryo, and its discharge, either as a complete cast or in shreds, explains the hæmorrhage. The patient usually recovers because intraperitoneal hæmorrhage is slight in cases of tubal abortion, or ceases spontaneously in cases of tubal rupture. All cases, however, do not run so characteristic a course. Quite commonly there is no history of a missed period, abortion or rupture occurring before the ovum has reached more than three or four weeks' development. Often, too, the events are not at all dramatic, the pain and faintness being so slight as to cause but momentary discomfort; the true nature of the case is only discovered later when the patient complains merely of pelvic pain and prolonged but slight bleeding from the uterus. Such cases may easily be mistaken for an incomplete uterine abortion, especially if a period has been missed, or if the patient has regarded herself as pregnant.

When severe intraperitoneal hæmorrhage occurs, the patient complains of constant agonizing pain, and has a progressive rise in the pulse-rate and increasing pallor. Areas of dullness may be detected in the abdomen, but no palpable mass; sometimes a fluid thrill can be elicited, and there is always great rigidity.

When a considerable retro-uterine hæmatocele

exists there are abdominal pain and rigidity, and a mass can be felt rising out of the pelvis more or less dull on percussion, according to the amount of intestine adherent in front of it. Rises of temperature due to blood absorption occur. Per vaginam the uterus is usually central, raised up out of the pelvis and pushed against the symphysis pubis, whilst behind it through the posterior fornix is felt a mass of a curious boggy consistence, indescribable, but characteristic of clotted blood. The vaginal mucous membrane is freely movable over the mass. If a hæmatocele becomes infected, the patient has severe toxic symptoms accompanied by high temperature, sweating, wasting and leucocytosis.

When the case is one of *peritubal hæmatocele* a mass can generally be felt reaching to the pelvic brim, and bimanually the uterus is felt pushed forwards and to one side, by a swelling having a hard or a boggy consistence. In all these cases there are usually no breast changes or other signs of pregnancy.

If the embryo continues to develop after abortion or rupture, there are as a rule no particular symptoms unless a secondary hæmorrhage occurs or the foetus dies. Generally the patient considers herself pregnant, and the condition is not diagnosed until some discomfort causes her to consult a doctor, or, full time having arrived, no normal labour ensues. A full-time foetus usually dies, and a mock labour occurs owing to the discharge of the decidua from the uterus, accompanied by hæmorrhage. Should the foetus die at any time during the gestation period, uterine hæmorrhage appears, and may be followed by cessation of all symptoms coincidently with the formation of a lithopædion. On the other hand, the sac may become infected and all the signs of a severe toxæmia supervene.

Diagnosis.—Cases with active intraperitoneal hæmorrhage are usually diagnosed easily. The sudden onset, the great abdominal pain, rigidity, increasing pallor, and gradually increasing pulse-rate make up a picture which can hardly be mistaken for any other lesion. If a period has been missed the diagnosis is more certain than when amenorrhœa is not present. In many cases with a dramatic onset, bleeding is small in amount, and consequently there is no progressive anæmia. A differential diagnosis may have to be made from *acute appendicitis*, *salpingo-oophoritis*, *ruptured gastric ulcer*, *ruptured ovarian cyst*, *torsion of the pedicle of an ovarian cyst*, *ectopic*

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from *intestinal, renal, or hepatic colic*. The missing of a period is a valuable diagnostic sign in such cases; when it is not present the diagnosis is correspondingly difficult, and cannot always be made correctly. There are a few points to be remembered. In acute appendicitis the pain is usually most acute at McBurney's spot, whereas in intraperitoneal bleeding it is not localized; in salpingo-oöphoritis pain is usually bilateral; in ruptured gastric ulcer it is more pronounced in the upper part of the abdomen. Pain is generalized in the lower part of the abdomen in rupture or torsion of an ovarian cyst. At the moment of rupture or abortion of a tubal gestation, and for some hours afterwards, there is no distinct pelvic mass, whereas in salpingo-oöphoritis there are bilateral masses, and in torsion of an ovarian cyst there may be quite a large tumour. In acute appendicitis no mass is felt as a rule, until the disease has lasted long enough for a localized abscess to form. Tubal gestation, ruptured gastric ulcer, and rupture or torsion of an ovarian cyst, are accompanied by shock, so that pallor may be common to all. In ruptured tubal gestation it increases rapidly, and the pulse-rate rises in frequency, if taken at ten-minute intervals. After the initial shock in the other lesions the pallor passes off, and the pulse-rate falls for a time. Only tubal gestation is associated with amenorrhoea, and is always accompanied by uterine bleeding sooner or later—usually very soon after the sudden onset of acute symptoms. In colic of various kinds there is, as a rule, localized pain which is intermittent and may be accompanied by vomiting, the pulse-rate being variable. Abdominal rigidity is unusual, but symptoms of shock may be present in severe cases. As a rule, colic is not easily mistaken for an "acute abdomen," under which term extra-uterine gestation and its accidents must be included.

When the primary acute symptoms have passed off and blood has clotted in the pelvis, diagnosis is usually easier. The presence of a pelvic mass, which is not uterine, in a person who has missed a period, and who gives a history of acute pain and shock some days or weeks previously, can hardly point to any lesion other than a hæmatocele. Unfortunately, amenorrhoea is not constant, and the acute symptoms may be of very short duration. A large retro-uterine hæmatocele may have to be distinguished from a *twisted ovarian cyst*; the history may be important, uterine hæmor-

rhage may have a bearing on the diagnosis, and usually the consistence of the two swellings is different. The way in which a hæmatocele pushes the uterus against the pubes is very characteristic, and rarely happens with an ovarian cyst. In the case of a small peritubal hæmatocele there may be considerable difficulty in deciding whether the uterus is separate from the pelvic mass—it may be enveloped in the bloodclot. More commonly the small hæmatocele has to be distinguished from an inflammatory mass due to *salpingo-oöphoritis*. The latter is usually bilateral, the former always unilateral. The uterus is displaced in hæmatocele, is usually central in salpingo-oöphoritis. The boggy feel of a hæmatocele contrasts with the hard inflammatory masses of salpingo-oöphoritis; and the vaginal mucous membrane is movable under a hæmatocele, but may be fixed in the case of inflammatory masses.

Treatment.—The treatment adopted depends on the clinical findings. In general, it may be said that most cases of tubal gestation require laparotomy, which gives very satisfactory results. If a tubal gestation can be diagnosed before abortion or rupture has occurred—a very uncommon occurrence—the affected tube should at once be removed, preserving the ovary. If abortion or rupture takes place immediate laparotomy is required, especially in the latter event, owing to the risk of severe or fatal intraperitoneal bleeding. As the surroundings of the patient are not always favourable to an operation, the question of removing her to a hospital or a nursing home has always to be faced. In most cases this can be done, but there are other cases in which it would be highly dangerous. The deciding factor will be the patient's general condition, and particularly the rate of the pulse. If the pulse-rate is not too rapid and is stationary, the inference will usually be that active bleeding has ceased. If the pulse-rate is steadily rising when counted at intervals of a few minutes, bleeding is still going on; removal of the patient in these circumstances would be a most hazardous proceeding, and the risk of an immediate operation, whatever the surroundings, would be less. In such cases the principle must be to open the abdomen, draw out the affected tube, and remove it after clamping the infundibulopelvic ligament and the uterine end of the tube and mesosalpinx. Then as much blood and clot as possible must be scooped out of the abdomen with the hand and swabs, the clamped

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broad ligament ligatured and the abdomen closed.

Subcutaneous saline infusion should be commenced in both axillæ before beginning the operation, and carried on until four or five pints have been infused. In a very severe case intravenous infusion should be resorted to, not more than three pints being run into the vein as a rule.

The anæsthetic chosen is a matter of importance, the best being gas and oxygen with novocain infiltration of the abdominal wall in three layers. Failing gas and oxygen, open ether anæsthesia, preceded by a hypodermic injection of morphia and atropine, answers very well.

There is always a considerable, and sometimes prolonged pyrexia after these operations.

If active bleeding has ceased, the operation is not so urgent, and arrangements can be made for placing the patient in the most favourable surroundings. When a definite hæmatocele has formed, as a result of abortion or of tubal rupture, removal of the blood mass and of the damaged tube by laparotomy is the best treatment, and saves a long period of invalidism. It must, however, be admitted that a number of such cases recover spontaneously, the blood-clot being slowly absorbed; but the results cannot be regarded as satisfactory to the patient, for she has a long period of invalidism, and must be left with a large number of adhesions in the pelvis.

In those cases in which the embryo continues to develop after rupture or abortion has occurred, it is clear that a satisfactory termination cannot be looked for by expectant methods; the fœtus and placenta can only be removed by operation. With regard to the best time for operation, it may be said that up to the sixth month immediate laparotomy should be undertaken. After that time, as is generally agreed, it is best to wait until at least a month after the fœtus has died, because of the danger of hæmorrhage when the placenta is separated for removal; this is always very severe, unless the maternal vessels can be ligatured beforehand. If the fœtus has been dead some time, the placental circulation to a great extent ceases, and the risk is then not so great, although it remains considerable. Further, there is no justification for operating in the interests of the fœtus, because extra-uterine fœtuses are often malformed, and usually have a poor expectation of life. There are exceptions to this rule, but they cannot be

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considered to weigh against the interests of the mother. Within limits, the longer the time which is allowed to elapse after the death of the fœtus, the better is the outlook for the mother, provided that the sac has not in the meantime become infected. The operation includes opening the abdomen, opening the sac, removal of the fœtus, and removal of the placenta when possible without undue risk. Whatever method is used the operation at this stage is highly dangerous.

THOS. G. STEVENS.

PREGNANCY, FIBROIDS COMPLICATING (see UTERUS, NEW GROWTHS OF).

PREGNANCY, INSANITY OF (see PUPERAL INSANITIES).

PREGNANCY, OVARIAN CYSTS COMPLICATING (see OVARIAN CYSTS).

PREGNANCY, MOLAR.—A mole is an ovum which has become destroyed by pathological changes affecting its coverings during the early months of gestation. It may occur both in uterine and extra-uterine pregnancies, and is sometimes called a *false conception* or *missed abortion*.

Moles are of two kinds—Blood Moles and Vesicular Moles.

1. **A Blood Mole** (*syn.* Carneous or Fleshy Mole) is due in some cases to traumatism, syphilis, or toxæmia. Abnormal activity of the trophoblast causes it to penetrate into the uterine wall and open up vessels so that hæmorrhage results and leads to partial or complete separation of the ovum. When the decidua is unhealthy it may undergo hyperplasia, and by enveloping the ovum cause hæmorrhages between the embryonic membranes and the decidua. If the mole is extruded from the uterus just after hæmorrhage has occurred, it is termed an *apoplectic ovum*.

Pathology.—The ovum becomes hæmorrhagic before formation of the placenta has taken place, and thus the chorio-decidual space becomes filled with blood from the maternal vessels and the embryonic chorionic villi are destroyed. The hæmorrhagic process takes place slowly and continuously, or is repeated at intervals and at various situations in the ovum. The amnion, being more elastic, may escape destruction. The embryo dies and becomes absorbed or shrivelled; traces of its cord, having a cystic appearance, may be found. The amniotic cavity frequently shows

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irregularities of its surface due to the unequal distribution of the effused blood around it. If the intradecidual hæmorrhage is considerable, the ovum becomes detached from the uterine wall and abortion occurs, but if there is still some vascular connexion the ovum may remain inert within the uterine cavity for weeks or months, during which the fluid portion of the blood is usually absorbed, leaving a firm mass which is seen to be laminated on section. On microscopic examination degenerated villi may be seen in the bloodclot.

Clinical features.—The patient shows the signs of early pregnancy. When the ovum dies there are usually some symptoms of threatened abortion, such as pain, hæmorrhage, or reddish-brown discharge. If these symptoms cease and the ovum is not expelled, the uterus may remain at the same size and menstruation be in abeyance. The ovum may be cast off later as a missed abortion.

The **diagnosis** is difficult, as the symptoms are indefinite; only expulsion of the mole makes the condition certain.

The **treatment** is that of threatened abortion. Leave the patient alone unless hæmorrhage continues or the discharge becomes offensive, when dilatation of the cervix and emptying of the uterus is advisable.

2. **Vesicular Mole** (*syn.* Hydatidiform Mole, Cystic Degeneration of the Chorion).—Cystic degeneration of the chorionic villi in the first half of pregnancy, resulting in the ovum being covered by masses of irregular-sized clusters of grape-like bodies. The degeneration may also occur in the placenta in the later months of pregnancy, and it has been found in extra-uterine pregnancy. It may affect only part of the ovum, and pregnancy may continue, but this is rare. Multiparæ between the ages of 30 and 40 are especially liable, and have been known to be affected in several pregnancies.

The **etiology** is obscure, but the condition may be due to some morbid change in the decidua affecting the chorionic villi. That it is an embryonic disease is shown by the fact that in twin pregnancies only one ovum may be diseased.

Pathology.—The stalks of the vesicles and the vesicles themselves, which are pale, translucent, and contain thin fluid without mucin,

are formed from the villi. There is irregular proliferation of both layers of the epithelium covering the villi, with degeneration and œdema of the stroma. The epithelium may penetrate the uterine wall and even perforate it unless the defensive round-celled infiltration keeps it in check. As a rule the embryo dies, becomes absorbed and mummified. Vesicular mole is sometimes associated with undue activity of the ovary and with lutein cysts.

Clinical features.—If only part of the ovum is affected and pregnancy continues, there are no symptoms. If the disease is extensive, irregular hæmorrhage occurs about the third or fourth month of pregnancy with enlargement of the uterus out of proportion to the duration of pregnancy. This increase in size of the uterus is sometimes rapid.

There is a thin, watery, brown discharge, but severe pain is absent unless abortion takes place. The presence of vesicles in the discharge is pathognomonic of the disease. Symptoms of toxæmia and pain on pressure over the uterus may be added. Spontaneous abortion may occur about the fifth or sixth month.

The **diagnosis** is uncertain unless vesicles are observed in the discharge. The general signs of pregnancy are present, but fetal parts or heart-sounds are not to be made out, unless in a twin pregnancy. Sometimes the mass can be felt within the cervical canal. If hæmorrhage is intermittent and the uterus unduly enlarged, the condition is difficult to diagnose from placenta prævia in some cases where the history is vague. Death may take place from hæmorrhage, sepsis, or malignant degeneration of the uterine contents.

Treatment.—If no diagnosis can be made, the patient must be closely observed, but nothing should be done until there is a clear indication. If the diagnosis is established, the uterus must be emptied immediately by dilating the cervix and removing the mole with the hand, taking care not to damage or perforate the uterine wall. A curette should not be used, as the uterine wall may already be invaded by the growth. Douche and pack if hæmorrhage is troublesome. If hæmorrhage recurs, explore the uterus and examine microscopically the tissue removed.

A. LOUISE McILROY.

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